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Table of Contents.

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	Page.
ORIGINAL ARTICLES—	
Malaria in the South-West Pacific, with Special Reference to its Chemotherapeutic Control, by N. Hamilton Fairley, C.B.E., F.R.S.	145
Deficiency Diseases, by B. L. W. Clarke	162
REVIEWS—	
Medical Treatment	166
Traumatic Conditions of the Upper Extremity	166
LEADING ARTICLES—	
The National Health Service Bill of Great Britain	167
CURRENT COMMENT—	
Poisoning with Ethylene Glycol	168
Lipoid Pneumonia	168
Non-Specific Urethritis and Reiter's Disease	169
ABSTRACTS FROM MEDICAL LITERATURE—	
Ophthalmology	170
Oto-Rhino-Laryngology	171
BIBLIOGRAPHY OF SCIENTIFIC AND INDUSTRIAL REPORTS—	
The Results of War-Time Research	172
BRITISH MEDICAL ASSOCIATION NEWS—	
Scientific	174
Notice	175
MEDICAL SOCIETIES—	
The Medical Sciences Club of South Australia	175
NAVAL, MILITARY AND AIR FORCE—	
Appointments	175
POST-GRADUATE WORK—	
The Melbourne Permanent Post-Graduate Committee	176
CORRESPONDENCE—	
A Method for the Removal of Tonsils and Adenoids under Local Anæsthesia with the Patient in the Recumbent Position	177
The Red Cross Rehabilitation Farm, "Gilbulla"	177
The National Health Service Bill of Great Britain	177
Spinal Anæsthesia and Chloroform: A Comparison of Mortality	178
Congenital Fibrocystic Disease of the Pancreas	178
PUBLIC HEALTH—	
Cancer Survey in New South Wales	178
AUSTRALIAN MEDICAL BOARD PROCEEDINGS—	
South Australia	179
Queensland	179
RESEARCH—	
Wyeth Fellowship in Clinical Medicine	179
OBITUARY—	
Harold Whitridge Davies	179
Leslie Gordon Davidson	180
Vera Scantlebury-Brown	180
NOMINATIONS AND ELECTIONS	180
THE FEDERAL MEDICAL WAR RELIEF FUND	180
NOTICE	180
BOOKS RECEIVED	180
DIARY FOR THE MONTH	180
MEDICAL APPOINTMENTS: IMPORTANT NOTICE	180
EDITORIAL NOTICES	180

MALARIA IN THE SOUTH-WEST PACIFIC, WITH SPECIAL REFERENCE TO ITS CHEMO-THERAPEUTIC CONTROL.

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PART I.

GENERAL REVIEW: INCIDENCE IN THE AUSTRALIAN MILITARY FORCES (1942–1945).

EVERY campaign conducted by the Australian Military Forces in the South-West Pacific from the outbreak of war with Japan until April, 1944, when the conquest of the Huon Peninsula ended, was accompanied by a severe epidemic of malaria. In all instances a large portion of the forces committed was affected.

Up to June, 1943, approximately 25,000 patients suffering from malaria were recorded as admitted to medical units. From that date until January 31, 1945, some 67,172 individuals were recorded as having had malaria. They suffered 132,014 attacks of the disease and spent a total of 2,545,585 days in hospital. This enormous drain on man-power resources was particularly severe in operational units and formations which suffered by far the heaviest incidence. Deaths directly due to malaria were uncommon. This was not due to the mildness of infections, but to early and adequate treatment, especially the universal practice of giving quinine intravenously in all doubtful cases. The actual mortality rate from uncomplicated malaria in these campaigns has not exceeded 1 in 3,000.

Malaria in New Britain, New Guinea and Australia (1942-1944).

On the return of the Australian Imperial Force divisions from the Middle East in March, 1942, the first grave problem facing the medical services was malaria.

New Britain.

This problem was illustrated by the experience of 252 men of the 2/22 Battalion who had escaped from Rabaul at the end of January, 1942. Their medical officer carried enough quinine to treat malaria casualties for approximately one month. After this their supplies of quinine became exhausted and fifty men, that is approximately 20%, died from malignant tertian malaria during the next five weeks before they escaped from New Britain to New Guinea. Practically all the survivors were heavily infected with the dangerous malignant tertian malaria and the recurrent benign tertian malaria as well. The malignant tertian infections were readily cured (radical cure), but though the benign tertian attacks responded satisfactorily to standard quinine, "Atebrin" and "Plasmoquine" ("Q.A.P.")[1] treatment, the temperature subsiding and parasites disappearing from the blood, relapses followed frequently at short intervals (about six weeks) in most instances. Antimalarial drugs produced a clinical, not a radical, cure in infections with New Britain strains of *Plasmodium vivax*. This was unexpected, as "Q.A.P."

[1] *Director*, Brigadier N. Hamilton Fairley; *Commanding Officer and Senior Physician*, Lieutenant-Colonel C. R. Bickerton Blackburn; *Entomologist*, Major M. J. Mackerras; *Pathologist*, Major T. S. Gregory; *Physician*, Captain R. H. Black; *Assistant Entomologists*, Lieutenant T. H. Lemerle and Lieutenant Q. N. Ercole; *Assistant Pathologists*, Lieutenant K. G. Pope, Lieutenant S. R. Dunn, Lieutenant M. S. A. Swan and Lieutenant T. A. F. Akhurst; *Officer Commanding Field Section*, Major I. C. MacDonald; *Pathologist, Field Section*, Major J. I. Tonge.

[1] Standard "Q.A.P." treatment in the Middle East consisted of quinine sulphate 30 grains daily for three days, "Atebrin" 0·3 gramme daily for five days and "Plasmoquine" (as base) 0·03 gramme daily for five days.

treatment had produced radical cure in approximately 70% of cases of vivax malaria contracted in the Middle East. This led to the adoption of another standard "Q.A.P." treatment for the South-West Pacific,[1] followed by a maintenance course of "Atebrin" (0·1 gramme daily for six weeks).

The salutary experience of the 2/22nd Battalion brought home the grim fact that forces exposed to infection when fighting in these highly malarious jungle areas in the South-West Pacific would inevitably be destroyed if they did not possess adequate supplies of quinine or other effective antimalaria drugs.

Port Moresby Area.

In June, 1942, Brigadier N. Hamilton Fairley, Director of Medicine, and Lieutenant-Colonel I. M. Mackerras, Director of Entomology, Land Headquarters, went to New Guinea to report on the tropical disease situation there. They found that 50% of troops in the dry Moresby area, which was regarded as only mildly malarious, were already infected with malaria. From the high incidence of malaria in natives at Milne Bay and Buna where fighting was anticipated, it was obvious the malaria hazard would far exceed that of Moresby. A medical appreciation was immediately sent by the Director-General of Medical Services to the Commander-in-Chief, General Sir Thomas Blamey, on the grave nature of the malaria casualties which would follow fighting in these areas, and appropriate recommendations were made.

Cairns.

To add to the general difficulties in June, July and August, there was an epidemic of benign tertian malaria at Cairns involving over 600 civilians. Up to this time wild anopheline mosquitoes infected with malaria had never been identified on the mainland of Australia, so the mosquito vector or vectors were unknown. As Australian troops were garrisoning the Cairns area at this time and as it was of vital military importance to prevent Allied bases in Northern Australia from becoming malarious, the Director-General of Medical Services requested the Director-General, Commonwealth Department of Health, to make available the services of Dr. G. A. M. Heydon, of the school of Public Health and Tropical Medicine, Sydney, who had previously found *Anopheles punctulatus* to be the vector in New Britain, in order to determine which species of anopheline mosquitoes was responsible for the Cairns epidemic. This was done. As the result of the researches undertaken by Dr. G. A. M. Heydon in conjunction with military entomologists at Cairns, it was proved that *Anopheles punctulatus moluccensis* was the vector. This discovery was of considerable practical importance, as it enabled rational measures to be instituted in adult mosquito and larvæ control based on the bionomics of the mosquito, and clarified the whole position regarding malaria transmission in tropical Australia.

This species of mosquito is not found in Australia below 30° south latitude, and for this reason epidemic malaria is only to be anticipated north of this level. Later Major F. H. S. Roberts proved that another anopheline mosquito, *Anopheles annulipes*, is an effective carrier experimentally and is capable of causing small outbreaks of malaria. Though it has a widespread geographical distribution in Australia, it fortunately is only of minor importance.

The Struggle for Antimalaria Supplies (1942–1943).

At this time, the stocks of antimalaria drugs held in Australia were inadequate for a tropical campaign. There was a limited supply of quinine and little "Atebrin". Mosquito nets were scarce in New Guinea and of inferior quality with such a poor air-flow that troops experienced great difficulty in sleeping under them in the humid tropics. Antimalaria equipment generally was in short supply. There was no satisfactory mosquito repellent. The urgent necessity for obtaining the highest priorities for all essential antimalarial supplies was stressed by the Director-General of Medical Services and the need for greatly increasing the antimalaria units and trained personnel emphasized.

Quinine.—Java produced 90% of the world's quinine supply, and while Advanced Headquarters, Australian Imperial Force was in Java in January and February, 1942, every effort was made to ensure that quinine supplies from there reached Australia. Approximately 100 tons of quinine was paid for in Java by the Australian Government early in February, but, owing to loss by enemy action, the inevitable difficulties facing a country on the verge of invasion and possibly fifth column activity, none reached Australia.

In 1942 the quinine reserve in the United Kingdom and United States of America approximated to about one year's normal world consumption. As it takes about seven years before a really satisfactory yield of quinine alkaloids is obtained from the bark of newly planted cinchona trees, it was evident that normal replacement of quinine stocks could not be maintained during the war, and every possible effort to conserve existing supplies would have to be made.

"Atebrin."—Following the failure to land quinine from Java, a large Australian order for "Atebrin" was placed in Washington, and it is of interest to note that it was the first military demand made for this drug on a large scale in the United States of America. In Australia the local manufacture of "Atebrin" was out of the question, as it would have been necessary at this time to import twenty tons of raw chemicals for every ton of the finished product.

"Atebrin" had been discovered by German chemists in 1931; patent rights were taken out, and its method of manufacture was kept secret by Germany. Russian chemists synthesized "Atebrin" a year or two later, but at the beginning of the war, except for small quantities manufactured in Russia and France, the whole of the world supplies of "Atebrin" still came from Germany. It was not until after the fall of Java that the United Kingdom and later the United States of America succeeded in manufacturing this drug.

In September, 1942, the Commander-in-Chief, General Sir Thomas Blamey, sent a mission to Washington and London to impress on the appropriate authorities the grave nature of the malaria problem in the South-West Pacific Area, the need for restricting the use of quinine to malaria, and the urgent necessity for the mass production of "Atebrin" and other essential antimalarial supplies. The mission included Dr. Adrien Albert, a leading Australian research chemist, who had special knowledge of the synthesis of "Atebrin". From all aspects this mission was successful. Quinine stocks were temporarily augmented by consignments from Great Britain, and certain quantities of "Atebrin" were sent from the United States of America.

Standard bobbinet was not available, but specifications for a new Nottingham type of mosquito netting were laid down; this later became a standard type for the British Army as well as for the Australian Armed Forces in the South-West Pacific Area. Dimethyl phthalate was advised as the most suitable mosquito repellent for the South-West Pacific, as it could be manufactured in Australia, was non-toxic when applied to the skin, and had been found by American entomologists to be effective against both mosquitoes and mites. Its efficacy against *Anopheles punctulatus* was demonstrated by Captain D. G. Waterhouse and against local mites by Major R. N. McCulloch. Later, in a most important series of field experiments in Australia and New Guinea McCulloch showed the superiority of dibutyl phthalate as a mite larvicide—its particular virtue when applied to clothing being that it afforded more prolonged protection, not being washed out so readily.

Many enormous claims now began to be made upon the limited production of "Atebrin" in the United States of America and the United Kingdom, and by March, 1943, the stock in hand in Australia could be measured in terms of a few weeks' supply. At the instance of Sir Alan Newton, Chairman of the Medical Equipment Control Com-

[1] Standard "Q.A.P." treatment for the South-West Pacific consisted of quinine sulphate 30 grains daily for three days, "Atebrin" in a daily dosage of 0·6, 0·5, 0·4, 0·3 and 0·2 gramme for five consecutive days, followed by "Plasmoquine" (as base) 0·03 gramme daily and quinine sulphate 15 grains daily for five consecutive days. This was followed by a maintenance course of "Atebrin" (0·1 gramme daily) for six weeks.

mittee, arrangements were made to send to the United States of America and the United Kingdom a second mission, concerned with the supply problems surrounding "Atebrin" and other essential medical items. This consisted of Mr. B. Egan, of the Division of Import Procurement, and Colonel C. Wallace Ross, Officer-in-Charge of Army Medical Supplies. After weeks of negotiation, the special needs of the South-West Pacific were recognized, and the flow of supplies recommenced. At the time the army held a very few days' supply of "Atebrin", and several consignments had to be flown from the United States of America and the United Kingdom to Australia. There was no actual break in supply, however.

Milne Bay and Buna-Gona Campaigns.

In the meantime, the Milne Bay and Buna-Gona campaigns had been successfully concluded, but the ratio of malaria casualties in each campaign had greatly exceeded battle casualties. In most instances troops were infected with both malignant tertian and benign tertian malaria which broke through despite the fact that they were taking, or had been advised to take, ten grains of quinine daily for prophylactic purposes. In all past military campaigns in the tropics, quinine had been the standard drug used for the control of malaria in the field. It was uncertain at this time whether its failure in New Guinea was due to failure of the drug, to irregular taking of quinine during the stress and strain of jungle warfare, or to both these factors.

The campaigns of late 1942 and early 1943 (Milne Bay, Kokoda, Buna and Wau) gave rise to thousands of cases of malaria, many of them severe. The Milne Bay operations took place in August-September, 1942, lasting about two weeks. Although malaria casualties were heavy, they did not greatly impede the course of the campaign. Reinforcement of the force in September and October, together with an intense multiplication of the malaria vector (*Anopheles punctulatus*), laid the foundation for a serious epidemic which, in November and December, nearly immobilized the Milne force. The maximum incidence occurred in the week ending December 25, 1942, when 1,083 patients were admitted to hospital out of a total strength of 12,000 men, giving a case rate of 82 per thousand per week or 4,264 per thousand *per annum*. The average rate for ten weeks prior to this peak was 33 per thousand per week or 1,720 per thousand *per annum*. A vigorous antimalaria campaign began in December. The case incidence fell rapidly and was only 8·2 per thousand per week by March 6, 1943. In a six months' period (October 23, 1942, to April 24, 1943) 8,099 cases of malaria were reported at Milne Bay, an overall rate of 26 per thousand per week or 1,350 per thousand *per annum*.

Meanwhile an arduous campaign was being fought in central Papua. After heavy fighting in the Owen Stanley Ranges, Australian troops encircled a Japanese force at Buna-Gona on the north coast of the island. Although little malaria had been contracted during the passage of the mountains, the troops were thoroughly exhausted and underfed. They were not equipped to protect themselves against malaria. Consequently a huge outbreak occurred in the course of three months' heavy fighting in the coastal swamp. Accurate figures of the incidence of malaria are not available, mainly because only seriously ill men were evacuated beyond regimental aid posts. During the period October 23, 1942, to April 24, 1943, 11,638 patients were admitted to medical units mostly from troops operating at Buna and Wau. The overall rate for operational troops was 22·5 per thousand per week, with the maximum incidence occurring in the last week of January, 1943, when 951 cases were reported equalling 48 per thousand per week or 2,496 per thousand *per annum*. This was the period of heaviest fighting at Buna.

A total of 20,272 cases of malaria was reported in New Guinea during the above six months' period. The overall case rate (including thousands of men at Port Moresby which was relatively well controlled) was 670 per thousand *per annum*.

It is emphasized that the full effects of this epidemic cannot be assessed from these figures. Hyperinfections were common. Thousands of men received brief courses of treatment in their unit lines. Thousands more treated themselves. The whole operation force was malarialized with very few exceptions. On their return to Australia in January-March, 1943, almost all of the men engaged at Buna had one or more relapses of benign tertian malaria. Table I gives a general indication of the scale of the relapse problem.

TABLE I.

Incidence of Repeated Malaria Attacks in the Australian Military Forces, June, 1943, to January, 1945.

Attack Number.	Number of Persons.	Percentage.
1	24,924	37·1
2	15,625	21·9
3	9,935	14·8
4	6,282	9·3
5	4,021	5·9
6	2,613	3·9
7 or more	3,772	7·1
Total ..	67,172	—

During the whole of the Papuan campaign antimalaria precautions were not carried out satisfactorily. There were grave deficiencies in mosquito nets, protective clothing, malaria control equipment and supplies, antimalaria drugs *et cetera*. The campaign began without any malaria control units being available on the island.

Quinine was the prescribed suppressive drug until January 15, 1943, when it was replaced by "Atebrin". There had been no supervision of drug parades and an inadequate realization of their importance. Probably only a small proportion of the troops took the amount ordered. It is improbable, however, that the change to "Atebrin" was responsible for the initial decline in rates early in 1943, but it must have been a contributory factor later by lowering the benign tertian relapse rate. The epidemic at Milne Bay was checked by other control measures, while the subsidence of casualties elsewhere occurred only when heavy fighting ceased and the infected troops were returned to Queensland.

By June, 1943, the total malaria rate in New Guinea was 7·6 per thousand per week or 395 per thousand *per annum*. This case incidence was occurring in troops established under static conditions in stabilized base areas. Officers and men alike still did not appreciate the efforts required to reduce the incidence of this disease under conditions of jungle fighting.

Preparations on the Mainland of Australia (1943).

On the mainland of Australia active preparations were made from every angle to meet the malaria menace in future jungle fighting.

As a result of the high rate of casualties from tropical diseases, especially malaria, and the difficulties of supplying reinforcements owing to manpower shortage, an urgent report was prepared for the War Cabinet by a special subcommittee surveying the whole field. This report left little doubt that if malaria casualties continued indefinitely at the high level attained in the Milne Bay and Buna-Gona campaigns, it would not be possible to maintain the requisite number of divisions at fighting strength.

About this time Sir Earle Page, who had been critical of the medical management of the malaria problem, was requested by the Prime Minister to report on the whole question. His subsequent report entirely vindicated the work of the Australian Army Medical Service in the diagnosis and treatment of malaria, and the general policy in the prevention and control of this disease adopted both in New Guinea and on the mainland of Australia.

In March, 1943, a personal advisory committee to General MacArthur on tropical medicine, hygiene and sanitation was established at General Headquarters, South-West Pacific Area, comprising Army, Air Force and Navy experts

of Australia and the United States of America, with Colonel M. J. Holmes, Australian Army Medical Corps, as the Chief Executive Officer. It was the function of this committee to advise the Supreme Allied Commander (*a*) on forward military planning from a disease viewpoint; (*b*) on drug supplies; (*c*) on every aspect of the prevention, control and cure of tropical disease in the Allied Forces; (*d*) on measures designed to prevent the introduction of such disease into Australia. The activities of this committee were little known, but they achieved a common allied policy in forward medical planning, in the cure, control and prevention of disease in the South-West Pacific, and in preventing the introduction of tropical disease by Allied troops into Australia.

In June, 1943, the Land Headquarters Medical Research Unit was established at Cairns to study the value of various antimalaria drugs in suppressing or preventing the development of overt malaria in experimentally infected volunteers in the tropics. No adequate experimental centre for critical investigations on human volunteers existed in any Allied country, and it was felt imperative that the Australian Army should be in a position to assess rapidly the value of new synthetic antimalarial drugs from this viewpoint. For this reason the Commander-in-Chief, General Sir Thomas Blamey, on the advice of the Director-General of Medical Services, decided to form such a research unit, under the direction of the Director of Medicine. The first three drugs tested were sulphamerazine, quinine and "Atebrin". It was found that though certain sulphonamides (one gramme daily) were effective in suppressing and even curing malignant tertian malaria, they were not true causal prophylactics and failed to suppress benign tertian malaria infections. Quinine (ten grains daily) completely failed to suppress heavy malignant tertian infections and sometimes failed to suppress benign tertian infections as well. It quickly became clear that "Atebrin" was a very effective suppressant of both benign tertian and malignant tertian malaria, but the full chemotherapeutic action of this drug had not been determined experimentally until the Huon Peninsula campaign was terminating.

During 1943 a considerable expansion of the antimalaria services took place under the directing influence of Colonel E. V. Keogh and Colonel E. Ford. The period March-September, 1943, was mainly occupied on the mainland of Australia with preparation for new offensive operations aimed at seizing the Huon Peninsula. Much attention was paid to the prevention of heavy malaria casualties. The equipment issued to troops was improved. Adequate protective clothing was issued to all ranks. Mosquito nets of improved pattern and material were available. The new mosquito repellent lotion, dimethyl phthalate, was manufactured in quantity. New malaria control units were raised and attached to operational forces as well as to base formations. A beginning was made with the incorporation of antimalaria training into general jungle warfare training. Orders specifying the commander's responsibilities for the administration of "Atebrin" to all troops were published.

The Huon Peninsula Campaign.

The Huon Peninsula campaign began in September, 1943, with a beach landing near Lae and air-borne landings in the Markham Valley. During the next five months the 7th Australian Division operated in the Markham and Ramu Valleys, 9th Australian Division with attached troops at Lae and in the Finschhafen area, and elements of the 5th Australian Division in the Salamaua and Lae areas. Contrary to expectation a formidable malaria epidemic occurred. Recorded malaria rates were of the same order as those experienced during the earlier campaigns in Papua, but the effect on the operational force was not nearly so great for several reasons. The general conditions and nutrition of troops were better and they were not subjected to such prolonged physical strain. "Atebrin" was plentiful, and though often irregularly taken, lessened the severity of the attacks. Evacuation to medical units was easier, so that sick men were not held in regimental aid posts. Finally, the method of recording malaria casualties had been greatly improved, so that case rates gave far more complete pictures of the malaria incidence than formerly.

The epidemic followed the usual course expected in a non-immune population in a hyperendemic area where adequate precautions are not practised. Beginning four to six weeks after the start of the operation, the rate rose sharply, reaching its maximum in the week ending November 26 (ten weeks after the start of action), when the overall rate for 30,000 operational troops was 55·3 per thousand per week or 2,875 per thousand *per annum*. In that week the total rate for the 7th Australian Division was 93 per thousand per week or 4,840 per thousand *per annum*. In this period the heaviest fighting and battle casualties occurred. Table III indicates the proportion of malaria casualties to total casualties.

TABLE II.

Australian Military Forces Malaria Casualties in Operational Areas (August 1, 1943, to April 30, 1944).

Type.	Number.	Percentage.
Benign tertian	6,545	30·8
Malignant tertian ..	10,830	50·9
Not confirmed	3,692	17·3
Others (mixed Quartan, Ovale)	216	1·0
Total cases	21,283	—

TABLE III.

The Proportion of Malaria Casualties to all Sickness Casualties and to Total Casualties including Battle Casualties.

Formation.	Total Malaria Casualties, September 4, 1943, to November 26, 1943, Expressed as Percentage of	
	(*a*) All Sickness Casualties.	(*b*) All Casualties (including Battle).
7th Australian Division (forward troops)	90%	83%
9th Australian Division (forward troops)	60%	44%

The rate for all troops stationed in New Guinea, including large forces in base areas, was 17·2 per thousand per week or 890 per thousand *per annum*. It is repeated that although these figures suggest an epidemic comparable in severity to those at Milne Bay, Buna *et cetera*, the total effect on the force was far less serious for the reasons given above. This was the unanimous opinion of observers who saw both campaigns. However, the position was so serious that early in December, 1943, after consultation with the Supreme Allied Commander, 7th Australian Division troops in the Ramu Valley were ordered to take 1·2 grammes of "Atebrin" every week. It had become clear that "Atebrin" was not being taken as prescribed, and this increased dosage was designed to check the epidemic by guaranteeing at least the consumption of an adequate suppressive dose, 0·6 to 0·7 gramme weekly, this amount of "Atebrin" having been shown by this time at the Land Headquarters Medical Research Unit, Cairns, to be adequate. Within a month of this change in dosage a considerable fall in the rate in the Ramu Valley occurred as is shown in Table IV.

This fall in incidence was not due solely to improved "Atebrin" discipline. The fighting was not so severe and the more heavily infected units were concentrating in rear areas for movement to Australia.

A new formation, 15th Australian Infantry Brigade, was flown into the area during the first week of January

to complete the drive up the valley to Madang. This brigade had spent five months during the latter half of 1943 fighting in the Wau-Salamaua area. It had evolved a complete organization for the enforcement of anti-malaria precautions which influenced subsequent orders issued on this subject in 1944 and 1945. Malaria prevention was made the direct responsibility of all unit and sub-unit commanders who were required to inform head-quarters daily that malaria precautions—including the wearing of protective clothing, use of mosquito nets, applications of repellent lotion and the taking of suppressive "Atebrin"—had been carried out. The Brigade Commander himself took a direct interest in the matter and set a high personal standard. In his own words: "The 'operations bogy' was not permitted to excuse slackness in carrying out malaria precautions."

TABLE IV.

Malaria Rates in Ramu Valley.

Period.	Average Rate per Thousand per Week.	Average Rate per Thousand *per Annum*.
December, 1943	43·7	2,270
January, 1944	14·3	740

In the first quarter of 1944 this brigade carried out extensive operations which resulted in the capture of Madang and Alexishafen. Its average malaria rate was 7·6 per thousand per week or 395 per thousand *per annum*, far the lowest figures achieved up to that time by infantry troops in action. Their rate was the same as that at Lae, which was then a base area with no operational hazard (including air raids) and with malaria control units at work.

Comment.—The malaria epidemic of this second New Guinea campaign was due to failure to carry out the prescribed precautions. The means were available, but were not properly used. The general failure of the force to take "Atebrin" regularly was the main cause of the high epidemic wave, though other aspects of both personal protection and field control were very unsatisfactory. For instance, the malaria control unit under command of the 7th Australian Division was not called into the operational area until nearly two months had elapsed. It was evident that if better results were to be achieved in the future, a big educational and training campaign would be necessary to stimulate the malaria consciousness, and particularly the "Atebrin" consciousness, of officers and men alike.

The opinion that a great reduction in malaria casualties could still be achieved was enormously strengthened by the experimental results obtained by the Land Head-quarters Medical Research Unit. By March, 1944, it had been conclusively proved that benign tertian malaria was completely suppressed by 0·4 gramme of "Atebrin" a week and that 0·6 to 0·7 gramme a week would not only suppress but cure malignant tertian malaria (New Guinea strains). In brief, what had formerly been only probable was now certain, the experimental proof being conclusive.

The Atherton Conference.

The Atherton Conference on Prevention of Disease in Tropical Warfare, which was held at Atherton on June 12 and 13, 1944, under the chairmanship of Lieutenant-General V. A. H. Sturdee, G.O.C., First Australian Army, was called as a direct result of the high casualties from tropical diseases in the Huon Peninsula campaign and the definite experimental findings regarding the chemo-therapeutic control of malaria with "Atebrin" at Cairns. The Adjutant-General, Major-General C. E. M. Lloyd, reported on hygiene and on the incidence of preventible tropical diseases in troops under command New Guinea Forces from September, 1943, to February 25, 1944. It was shown that with an average posted strength of 98,050 men there were 3,140 battle casualties and 47,534 evacuated sick during this period.

Brigadier N. Hamilton Fairley dealt with "Atebrin" suppression of malaria. It was shown that from a military viewpoint the implications of the Cairns experiments were far-reaching. If these experimental results were true, it followed that any force of non-immune troops who built up and maintained an adequate concentration of "Atebrin" in the blood throughout the period of exposure and for four weeks thereafter could fight in highly malarious country with an absolute minimum of malaria casualties. Since malignant tertian malaria was cured by this regimen, there should be no death rate from malaria and no blackwater fever. Carriers within the force would not exist, since sexual parasites did not appear in the blood provided one tablet of "Atebrin" (0·1 gramme) was taken every day with invariable regularity. When the force left the malarious area, returned to Australia and stopped taking "Atebrin", relapses of benign tertian malaria would be expected in a period of three to twelve weeks. The percentage of benign tertian relapses would depend on the efficacy of those measures of personal protection, such as the efficient use of mosquito net, correct clothing and the application of mosquito repellent lotion at appropriate times.

If antilarval measures and adult mosquito destruction had been efficiently carried out and if antimalaria discipline had been effective, the incidence of benign tertian malaria would be low; if there had been laxity the rate would be high.

Major J. C. English, malariologist, 1st Australian Corps, dealt very completely with the prevention of malaria in the field, and Major C. E. Cook detailed measures necessary for the control of intestinal diseases—dysentery and cholera. Major-General S. R. Burston terminated the discussion by pointing out to the divisional and brigade commanders that "the ball was now in their court".

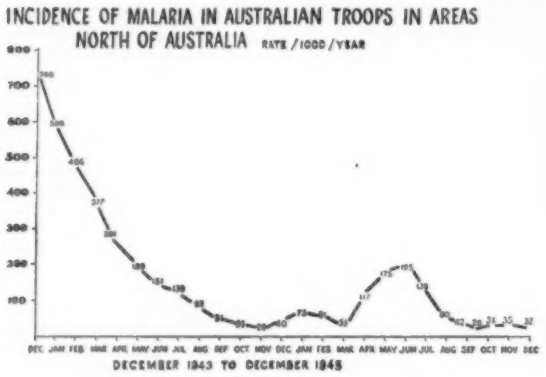
Lieutenant-General V. A. H. Sturdee, in closing the conference, said that there was no doubt that many officers had come to the conference with little enthusiasm, if not hostility. However, in a very short time the Director-General of Medical Services and his assistants had completely convinced all present of the seriousness of the position and of the need for the whole-hearted cooperation of all ranks. So successful had the conference been, especially on antimalarial measures, that commanders and staffs present now appeared to be desirous of pressing the medical service further even than the latter was prepared to go. It was most important that the vital information placed before the conference should be conveyed by the same lecturers to the maximum number of unit officers and senior non-commissioned officers on the tableland, in order to achieve fully effective measures against tropical diseases.

The Atherton conference was undoubtedly a turning point in the medical history of the war. An extensive educational and training campaign was immediately organized amongst all ranks for the operations planned to take place in late 1944 and 1945.

Concurrently, improved antimalarial discipline produced its effects in the garrison troops remaining in New Guinea. "Atebrin" discipline was tightened up, with the result that by September, 1944, the malaria rate in New Guinea was one per thousand per week or 52 per thousand *per annum*, and by November, 1944, 0·5 per thousand per week or 26 per thousand *per annum* (Graph I).

Projected operations were faced optimistically. The application of experimental results to field conditions had proved effective. All ranks appeared to be thoroughly educated, trained and equipped to protect themselves against malaria. The remarkable efficacy of "Atebrin" had been conclusively proved, and it was therefore anticipated that malaria casualties would in future not cause significant interference with operations.

Experience largely fulfilled this expectation. With one exception, namely, that of the 6th Division, the Cairns experiments were reproduced on a large scale under field conditions.



GRAPH I.

Malaria in the Australian Military Forces in the South-West Pacific (1944-1945).

The new round of actions began in November, 1944, continuing until the end of the war. D.D.T. was employed both as a larvicide and for adult destruction; its use enabled effective control to be established more rapidly than by other methods. Air spraying with D.D.T. was practised in all areas, but not on a large scale, except at Borneo and possibly Bougainville. During this period excellent work was done in malaria control by the malaria control units under the direction of Lieutenant-Colonel J. C. English and Major F. J. Fenner, but the main factor in the low malaria rate was undoubtedly attributable to the high standard of "Atebrin" discipline attained.

Location and Strength of the Forces Involved.

The areas concerned with the approximate total forces involved were:

1. *Aitape-Wewak.*—Twenty-three thousand men comprising a divisional group and a base sub-area. One of the three brigades concerned had no prior experience in malarious areas of the South-West Pacific.

2. *New Britain.*—Fifteen thousand men, including a division and a base sub-area. One brigade had no prior experience of malarious areas.

3. *Solomons.*—Fifty thousand men. Five brigades and a base sub-area were operating. One of these brigades had no earlier experience of malarious areas.

4. *Morotai-Borneo.*—Eighty-five thousand men. Two divisions, various base groups and numerous corps troops were involved. The major formations had prior experience of malarious areas. Operations did not begin until April, 1945.

5. Various base areas, on a reduced scale, were maintained in New Guinea, especially at Lae and Madang. The brigade group at Madang had been in New Guinea about one year. The total troops equalled 15,000.

In the various operational groups there were many new units and individuals who were entering malarious areas for the first time.

Malaria Risk in Different Operational Areas.

The malaria risk in all areas was high, though not necessarily uniform.

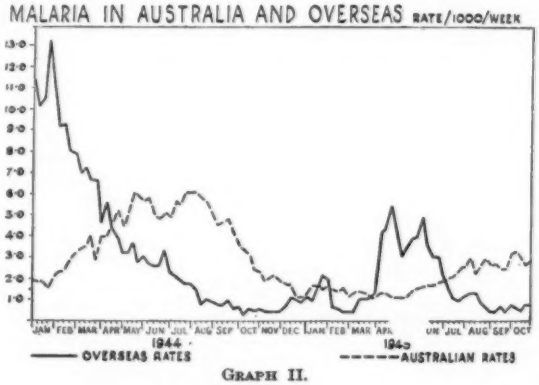
Earlier experience in New Guinea and New Britain indicated that the malaria hazard in these areas was very high.

In the Solomons native spleen rates were of the same order as those found in New Guinea. An indication of the malaria hazard in the Solomons was recently provided by the experience of Japanese prisoners of war concentrated in the Fauro Islands. Seven hundred and ninety-nine prisoners from Nauru, a non-malarious island, with their own medical service, were sent to Fauro on October 30 and 31, 1945, after a brief period of staging at Torokina. The first case of malaria occurred on November 8, and was followed by an epidemic involving almost every man. On November 26, four weeks after entering the malarious area, 530 patients were in hospital undergoing anti-malarial treatment. By December 5, thirty-five days after movement to Fauro, 212 deaths had occurred (26·5%). This experience was reminiscent of the 252 members of the 2/22 Australian Infantry Battalion escaping from Rabaul quoted earlier. Although these were special circumstances, in that these men were found in camps with heavily infected troops, their enormous death rate again indicated the fate awaiting any group of non-immunes not protected by "Atebrin" or other effective antimalaria suppressant and not adequately treated once overt malaria developed.

The coastal belt of Borneo and country extending up into the foothills were also highly malarious, except for those small areas in which pre-war industry and commerce necessitated some degree of malaria control. This control had been sometimes of good quality, but its condition after three years' occupation by the Japanese was doubtful. It is reasonable to describe all areas as hyperendemic.

Malaria Incidence.

The general incidence of malaria in the Australian Military Forces overseas is included in Graphs II and III and in Tables V and VI. The rise in the rates in the middle of 1945 resulted entirely from an outbreak in the 6th Australian Infantry Division in the Aitape-Wewak area



GRAPH II.

of New Guinea, which is considered in Part II. In New Guinea itself other than in the Aitape-Wewak area, very low rates were recorded. The monthly rates in 1945 varied from 16 to 52 per thousand *per annum*, being highest in February and lowest in November.

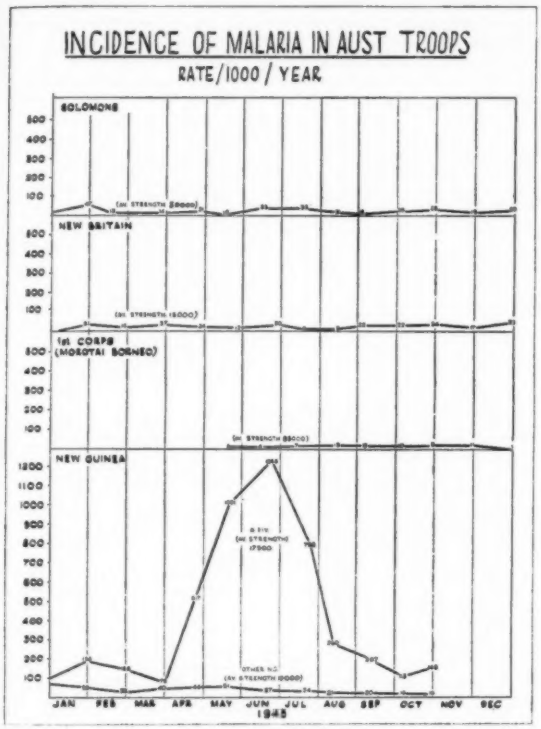
The malaria incidence in operational areas other than New Guinea (1944-1945) was insignificant from a military viewpoint.

Out of a total force of approximately 130,000 troops exposed for a period from approximately six to twelve months, there were only 1,256 attacks of malaria. Thus the combined malaria incidence was 0·97%. In the Solomons the incidence over a period of approximately eleven months was 2·2%, in New Britain over a similar period it was 1·8%, and in the Morotai-Borneo force over a period of approximately six months it was only 0·37%.

The type of malaria encountered in these three campaigns is summarized in Table VI.

It will be seen from a perusal of Table VI that of the 1,256 attacks of malaria, 476 were due to *Plasmodium falciparum*, 658 to *Plasmodium vivax* and one to *Plasmodium malariæ*. In sixteen instances both benign tertian and malignant tertian parasites were found. Diagnosis was made on clinical grounds in the absence of demonstrable parasites in 105 cases. The predominance of *Plasmodium vivax* is of considerable interest, as it indicates that less than four tablets of "Atebrin" were being taken by the 658 men developing overt benign tertian malaria.

In these campaigns the case rate rarely rose above one per thousand per week and was often considerably less. When any temporary rise occurred some breakdown

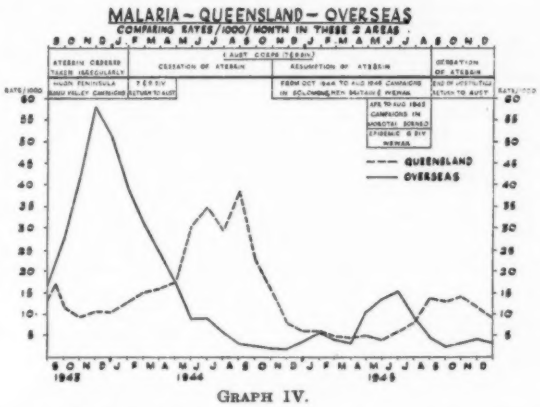


GRAPH III.

in "Atebrin" administration could often be detected. For instance, 34 cases occurred in each of two successive weeks at Bougainville. It was found that the cases were confined almost entirely to one brigade which admitted laxity in supervision of the units from which the patients came. Disciplinary action abruptly checked the outbreak which was not repeated in the heavy fighting which occurred subsequently in the campaign.

Malaria in Returned Troops after Ceasing to Take "Atebrin".

This remarkably low incidence of malaria in the bulk of overseas troops was in marked contrast to earlier outbreaks of the disease in New Guinea and to the rates seen in troops serving on the mainland of Australia where malaria transmission was not occurring and where troops had ceased to take suppressive "Atebrin" (unless for some specific reason they were specially ordered to do so). Graphs II and IV and accompanying table indicate the



GRAPH IV.

change which took place during the period under review. It will be seen that, following the return to Queensland of the 7th and 9th Australian Divisions and supporting troops from the Huon Peninsula campaign, rates in New Guinea fell rapidly to the low level indicated previously; rates in Queensland on the other hand rose sharply, after the returned troops ceased "Atebrin" suppression. The figures for Queensland are not based on the population at risk, that is, men who were exposed to infection, but on the total military population in the State at that particular time, including lines of communication troops *et cetera*. This massive benign tertian malaria wave may be summarized by comparing the cases and rates in the two areas for two periods of 24 and 28 weeks, the first being the period of operations in New Guinea and the second the

TABLE V.
Malaria Incidence in Operational Areas other than New Guinea (1944–1945).

Location.	Period of Observation.	Total Force at Risk.	Total Malaria Attacks.	Percentage Infected.
Solomons	October 27,1944, to October 5, 1945 (11 months, 8 days)	50,000	666	2·2
New Britain	October 27, 1944, to October 5, 1945 (11 months, 8 days)	15,000	277	1·8
Morotai-Borneo	April 13, 1945, to October 5, 1945 (5 months, 22 days)	85,000	313	0·37
Total	.. ..	130,000	1,256	0·97

TABLE VI.
Type of Malaria found in 1,256 Overt Attacks in Troops in the Solomons, New Britain, Morotai and Borneo.

Location of Force.	Malignant Tertian.	Benign Tertian.	Quartan.	Malignant Tertian and Benign Tertian.	Not Confirmed. (Clinical Diagnosis.)	Total Attacks.
Solomons	311	278	0	14	63	666
New Britain	45	200	0	1	31	277
Morotai-Borneo	120	180	1	1	11	313
Total	476	658	1	16	105	1,256

period of rest, leave and rehabilitation in Queensland where malaria is not endemic and where malaria transmission was not occurring.

Practically all cases were due to benign tertian relapsing malaria in Queensland. The table provides a general measure of the degree to which troops who, between October, 1944, and April, 1945, returned to fight hard campaigns, were infected in earlier operations.

The progressive tendency for the heaviest incidence of malaria to occur in formations resting in Australia may be followed in the graphs. With the exception of the 6th Australian Division, formations serving outside Australia, in areas with high malaria transmission, suffered very minor malaria casualties. The disease remained a constant potential menace, but at no time became an important cause of admission to hospital.

Sixth Australian Infantry Brigade.—Insufficient time has elapsed since the ending of the war to permit a complete survey into the extent to which returning troops have been infected with malaria. It is possible to say, however, that the incidence is high. In June and July, 1945, the 6th Australian Infantry Brigade returned to the mainland from New Britain for disbandment. It had spent twenty-one months in malarious areas, twelve of them in New Guinea (Buna and Lae) and nine in New Britain. In its first months at Buna it had heavy malaria

TABLE VII.

Comparison of the Incidence of Malaria in Troops[1] in New Guinea and Queensland.

Location.	September 10, 1943, to February 25, 1944.		February 25, 1944, to September 9, 1944.	
	Total Cases.	Average Rate per 1,000 per Week.	Total Cases.	Average Rate per 1,000 per Week.
All troops in New Guinea	29,653	9·6	6,698	3·0
All troops in Queensland ..	10,583	3·0	32,827	7·1

[1] Troops in New Guinea were on "Atebrin" suppression, whereas those in Queensland were not.

casualties. A great improvement in protective measures, particularly in "Atebrin" administration, then occurred. The result was that malaria rates fell sharply. In the year prior to the brigade's return to Australia the highest incidence rate was five cases per thousand per week in August, 1944. The average weekly rate for the whole year was well below one per thousand per week. It is reasonable to call this the "minimal malaria casualties" foreshadowed in General Routine Order A.404/1944.

Thus for twelve months the brigade showed no significant evidence of malarial infection. It became customary for critics to assert that New Britain was "not very malarious" in spite of tragic experiences in this same area in 1942. A severe outbreak of malaria (benign tertian) when "Atebrin" ceased to be taken after returning to the mainland completely dispelled this illusion.

The 14th/32nd and 19th Australian Infantry Battalions left New Britain in the first week of June, the 36th Australian Infantry Battalion one month later. Disbandment ended late in September. "Atebrin" was taken for one month after their departure from New Britain. Only odd cases of malaria were reported in this period. Beginning in each instance about six weeks after disembarkation an epidemic of benign tertian malaria took place, continuing throughout the eight-week period of disbandment.

There were two attacks of quartan malaria (*Plasmodium malariæ*), one mixed benign tertian and malignant tertian infection and one malignant tertian. The remaining 972 were all benign tertian malaria, *Plasmodium vivax* being demonstrated in the blood smears.

Unfortunately, it proved impossible to obtain a complete record of the malaria casualties owing to the disbandment of the brigade which thereby entirely lost its identity. It must be stressed that the figures do not give anything like a complete account of the real incidence of benign tertian malaria in the brigade group for the following reasons: (*a*) The strength was that on departure from New Britain and makes no allowance for the piecemeal disbandment which took place. (*b*) Queensland lines of communication

TABLE VIII.

The Incidence of Benign Tertian Malaria in Returned Troops of the 6th Australian Infantry Brigade after They Ceased Taking "Atebrin".

Battalion.	Strength.	Malaria Attacks.	Percentage.
14/32nd	729	292	40·0
19th	724	416	57·6
36th	729	268	36·8
Total	2,182	976	44·7

records only were used. (*c*) No allowance is made for personnel on leave, transfer *et cetera*. (*d*) The average period of observation after cessation of "Atebrin" was only eight weeks, far too short a time for all suppressed cases to have developed.

The highest rate recorded in any week was 125 per thousand. It is estimated that the ultimate benign tertian infection rate will be 70% or more.

This incident affords dramatic proof of the efficacy of "Atebrin" as a suppressant. A tremendous benign tertian malaria infection was unmasked within a few weeks following the cessation of 0·1 gramme of "Atebrin" daily. In addition, direct proof of the cure of malignant tertian malaria was provided. Only two men out of nearly one thousand diagnosed had *Plasmodium falciparum* parasites in their blood, although it is reasonable to assume that at least as much malignant tertian malaria as benign tertian was being transmitted in New Britain. In fact earlier experiences on this littoral would suggest unmistakably that malignant tertian is transmitted there at a greater rate than benign tertian malaria.

Summary.

Australian Military Forces experience in the suppression of malaria in the 1944-1945 period may be summarized thus:

1. A very high incidence of benign tertian malaria occurred amongst troops returning to Australia after the Huon Peninsula and Ramu Valley campaigns. At the same time a more responsible attitude towards the taking of "Atebrin" brought about a sharp decline in malaria rates amongst those troops remaining in New Guinea. Rates were experienced there conforming to the "minimal malaria casualties" suggested by experimental work.
2. Malaria ceased to be an operational problem in subsequent campaigns in the Solomons, New Britain and Borneo.
3. Benign tertian malaria became a problem on the mainland following cessation of the taking of "Atebrin" by troops returned there for rest and regrouping.
4. The cure of the vast majority of malignant tertian infections was effected by suppressive "Atebrin" without significant symptoms of malaria infection ever being manifest. As a result very few deaths or serious complications from malaria occurred.
5. The general health and condition of troops returning to Australia were greatly improved.
6. In only one area in New Guinea, namely, Aitape-Wewak, were these splendid results not obtained.

PART II.

MALARIA IN THE 6TH AUSTRALIAN DIVISION (AITAPE-WEWAK AREA).

In the course of this campaign along the New Guinea northern coastline extending from Aitape to Wewak, the 6th Australian Division suffered a severe outbreak of malaria—chiefly malignant tertian. To a lesser degree the 3rd Australian Base Sub-Area troops were also involved.

Cases were widespread, with the heaviest incidence in the 16th, 17th and 19th Brigades. The highest rate (113·2 per 1,000 per week) developed in the 16th Brigade in May, 1945. By September, one month after the end of the war, rates had fallen to a relatively low level.

Statistical Analysis of the Epidemic.

Malaria Incidence in the Aitape-Wewak Area.

It will be seen from Table IX that of a total force of 17,500 men of the 6th Australian Division, engaged in jungle fighting for a period of over eleven months in the Aitape-Wewak area, 4,838 individuals suffered from one or more attacks of malaria. Only four deaths from malaria were recorded during this campaign and none from blackwater fever.

TABLE IX.

Malaria Incidence in the Aitape-Wewak Area (October 20, 1944, to September 28, 1945).

Formation.	Total Force at Risk.	Total Individuals having One or More Attacks.	Total Attacks of Malaria.	Average Attack per Patient.
6th Australian Division.	17,500	4,838 (27·6%)	6,285	1·3
3rd Australian Base Sub-area.	5,500	539 (9·8%)	621	1·15
Total	23,000	5,377 (23·3%)	6,906	1·28

Some 27·6% of the total force developed overt malaria attacks. Judging by experience in past campaigns, it may be assumed that at least 90% of the force would have acquired malignant tertian or benign tertian infections or both at some time during this period. If so, then "Atebrin" was effective in suppressing major clinical symptoms in 62·4% over a period of exposure exceeding eleven months.

In the 3rd Australian Base Sub-Area, where conditions were more static, 539 troops out of 5,500 developed overt attacks, that is, 9·8%.

Type of Malaria.

Details in regard to the type of malaria are given in Table X. It will be seen that malignant tertian infections were most common; parasites of *Plasmodium falciparum* were demonstrated in 78·2% of the total cases (single and mixed infections). *Plasmodium vivax* was found in

TABLE X.

Type of Malaria Found in 6,906 Overt Attacks in the Aitape-Wewak Area (October 20, 1944, to September 28, 1945).

Formation.	Malignant Tertian Malaria.	Benign Tertian Malaria.	Malignant Tertian and Benign Tertian Malaria.	"Not Confirmed" (Clinical Diagnosis).	Total Attacks.
6th Australian Division.	4,779 (76·0%)	666 (10·6%)	135 (2·2%)	705 (11·2%)	6,285
3rd Australian Base Sub-area.	478 (76·9%)	75 (12·2%)	9 (1·4%)	59 (9·5%)	621
Total ..	5,257 (76·1%)	741 (10·7%)	144 (2·1%)	764 (11·1%)	6,906

12·8% (single and mixed infections). In 11·1% the diagnosis was not confirmed by the finding of parasites. This "not confirmed" group included clinical malaria and probable infections like dengue fever and other febrile conditions in which the medical officer felt that the patient should receive the benefit of the doubt and have antimalaria treatment without further delay.

Multiple Attacks.

It will be seen that the proportion of men suffering from multiple attacks was not high. In troops of the 6th Australian Division the average attack per patient was 1·3, and in the 3rd Australian Base Sub-Area troops 1·15 per patient over a period exceeding eleven months (Table IX). When it is remembered that this group included recrudescences and relapses as well as new infections, the rate was unexpectedly low and quite at variance with the prevalent medical opinion in the Aitape-Wewak area where recrudescences and relapses following treatment were generally regarded as very much more frequent. This point has considerable significance when the problem of "Atebrin"-resistant strains receives consideration. The actual frequency of attacks is given in Table XI.

TABLE XI.

Frequency of Attacks.

Attack.	Number of Attacks.	
	6th Australian Division.	3rd Australian Base Sub-area.
First	3,737	471
Second	829	56
Third	205	11
Fourth	60	0
Fifth	7	1
Sixth	0	0
Total	4,838	539

In the 6th Australian Division only 1,101 men (22·7% of those affected) had more than one attack, while in the 3rd Australian Base Sub-Area only 68 (12·6%) had more than one attack.

Further analysis of the cases classified according to the type of parasite causing the infection are given in Tables XII and XIII.

Comparison with the incidence of malaria in the 7th and 9th Australian Divisions during the Ramu Valley and Huon Peninsula campaigns (September, 1943, to March, 1944) indicated that the proportion of "subsequent attacks" after the first attack was approximately the same.

TABLE XII.

Malignant Tertian Malaria[1]; Incidence of Attacks.

Attack.	6th Australian Division.			3rd Australian Base Sub-Area.		
	Officers.	Other Ranks.	Total.	Officers.	Other Ranks.	Total.
First	108	3,370	3,478	8	415	423
Second	16	728	744	2	45	47
Third	5	153	158	0	9	9
Fourth	0	37	37	0	0	0
Fifth	0	6	6	0	1	1
Sixth or subsequent	0	0	0	0	0	0
Total ..	129	4,294	4,423	10	470	480

[1] Includes all cases diagnosed as "malignant tertian", "not confirmed" or "mixed".

For purpose of comparison, data concerning the incidence of multiple attacks are epitomized in Table XV.

The likelihood of contracting more than one attack of malaria at Wewak was thus not greater than the chance of so doing in earlier campaigns of lesser duration during which no "Atebrin"-refractory strains of parasites were suspected to exist. It would be expected that subsequent attacks of malignant tertian malaria particularly would be far commoner were such a strain of dominant importance at Wewak.

TABLE XIII.

Benign Tertian Malaria[1]; Incidence of Attacks.

Attack.	6th Australian Division.			3rd Australian Base Sub-Area.		
	Officers.	Other Ranks.	Total.	Officers.	Other Ranks.	Total.
First	11	692	703	0	75	75
Second	1	44	45	0	3	3
Third	0	3	3	0	0	0
Fourth or subsequent	0	0	0	0	0	0
Total ..	12	739	751	0	78	78

[1] Includes all cases diagnosed as "benign tertian" or "mixed".

TABLE XIV.

Malaria, 7th and 9th Australian Divisions, September 3, 1943, to January 7, 1944. (Operational Areas Only.) Incidence of Attacks.

Attack.	7th Australian Division.	9th Australian Division.
First	2,569	3,543
Second	812	746
Third	199	118
Fourth	19	34
Fifth	1	34
Sixth or subsequent ..	0	17
Total	3,600	4,492

TABLE XV.

Incidence of Multiple Attacks in 6th, 7th and 9th Australian Divisions.

Formation.	Total Individuals.	Number with More than One Attack.	Percentage.
6th Australian Division ..	4,838	1,101	22·7
7th Australian Division ..	3,600	1,021	28·3
9th Australian Division ..	4,492	959	21·3

It should be noted that the 6th Australian Division figures are derived from an experience lasting approximately eleven months, whilst those for the 7th and 9th Australian Divisions occurred during a period of four months. This makes the comparison more striking.

If it be conceded that all individuals at Aitape and Wewak with subsequent attacks of malignant tertian malaria suffered from recrudescences or relapses rather than reinfections, the percentage of malignant tertian sufferers likely to have the resistant strain would be only 21·3% (945 out of 4,423 individual sufferers). This, of course, is too high a figure, as a considerable number would be reinfected following discharge from hospital. It follows that considerably less than one-fifth of men admitted to medical units with malignant tertian malaria were infected with the relatively "Atebrin"-resistant strain which was later proved to be present in the area.

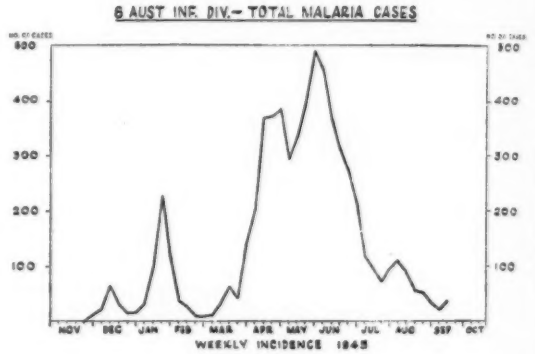
Thus the outbreak at Wewak was confined to approximately one-quarter of the divisional force and one-tenth of base troops. The frequency of subsequent attacks after the first was not high, particularly in view of the claim made by medical officers that relapses occurring quickly after the completion of standard treatment were frequent. This was particularly so, when it is remembered that an undetermined proportion of second attacks would inevitably have been due to reinfection.

Incidence of Malaria in Officers and Other Ranks.

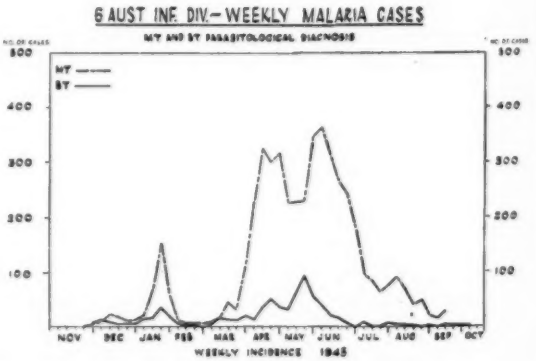
During the campaign only 137 (15%) of the officers serving with the 6th Australian Division were admitted to medical units with diagnosed malarial attacks. This means the incidence of malaria breakdown in officers was approximately one-half that of non-commissioned officers and men.

Course of the Epidemic.

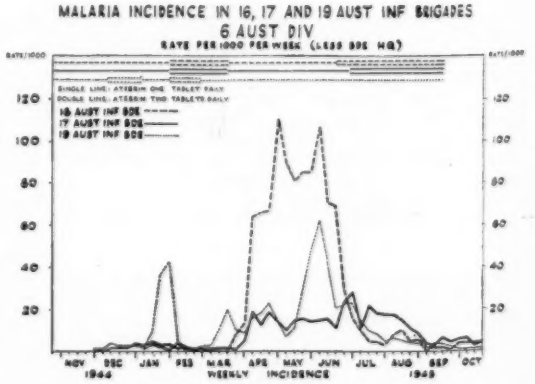
Graphs V and VI provide a general picture of the course of the epidemic in the 6th Australian Division. It is based on weekly cases (not rates). By comparison with Graph VII, which presents weekly rates in the three brigades, it will be found that the initial rise, occurring in January-February, 1945, was confined to the 19th Australian Infantry Brigade. This outbreak was heralded by an undue case incidence in the 2/8 Australian Field Company and the 2/10 Australian Cavalry (Commando) Squadron



GRAPH V.



GRAPH VI.



GRAPH VII.

belonging to the 19th Australian Infantry Brigade group. Investigation revealed administrative failures in these two units and disciplinary action was taken. As an emergency measure the whole brigade group was ordered to take 0·2 gramme of "Atebrin" daily from December 13, 1944, to January 4, 1945 (approximately three weeks). The brigade returned to Aitape to rest at the end of January, during which time rates rose rapidly, reaching 44·8 per thousand per week for the week ending February 2, 1945. It was decided, in the words of the Deputy Assistant Director of Hygiene of the 6th Australian Division, "to place the whole Division on two tablets to terminate the rising incidence in 19 Aust. Inf. Bde. group and avoid the possibility of a similar occurrence in the remainder of the Division". The 19th Australian Infantry Brigade reverted to one tablet at the end of February, but the 16th and 17th Brigades continued to take two tablets daily until March 22. In this period the 17th Brigade moved south into the Torricelli Mountains and operated throughout the rest of the campaign in these mountains and the Prince Alexander Range with a battalion resting at Aitape. The generally lower rates for this brigade may have resulted from a lower rate of transmission in these areas.

The subsequent course of the epidemic is clearly shown in Graphs V, VI and VII. The highest rates reached were in the 16th Brigade, which suffered 113 per thousand per week in the week ending May 4 and 110 per thousand per week in the week ending June 8. In the latter week, the 19th Brigade reached its peak of 65 per thousand per week. The 17th Brigade reached 20 per thousand per week during the week ending April 20 and 30 per thousand per week during the week ending July 6, with "moderate" rates of 10 to 15 per thousand per week in the intervening period.

The relationship of the epidemic curve in the three brigades to the "Atebrin" dosage as indicated in Graph VII is of considerable interest. At the time of the initial outbreak in January-February the fall in the 19th Australian Infantry Brigade was ascribed to the increase in dosage. When the epidemic began to decline in the 16th Australian Infantry Brigade in July-August, this fall was also attributed to increased dosage, though it will be seen that the fall had begun before the dose was increased. The 19th Australian Infantry Brigade declined almost *pari passu* with the 16th Australian Infantry Brigade, although no increased dosage was prescribed. Finally, the 17th Australian Infantry Brigade underwent no reduction in rates for many weeks after the order was issued that two tablets daily be taken.

Malaria Incidence in the 8th Australian Infantry Brigade.

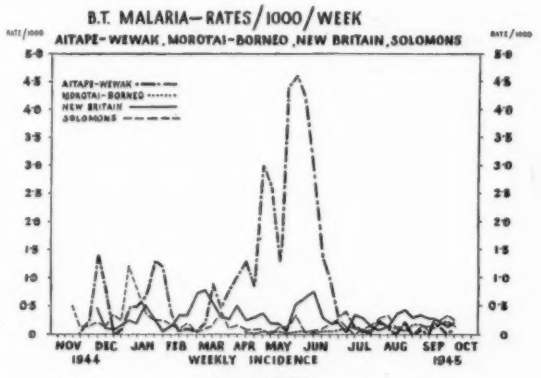
During the first half of July a part of the 8th Australian Infantry Brigade was moved into the Wewak area from Madang—Alexishafen—Hansa Bay. This brigade landed at Finschhafen on January 22, 1944. It participated in two long marches up the coast, the first in February, 1944, from Sio to Saidor, and the second in April from Alexishafen to Hansa Bay. In this period the 30th Australian Infantry Battalion suffered 275 malaria casualties and the 35th Australian Infantry Battalion 110. In both instances almost all the cases occurred in the first few months of 1944. During the year prior to their move to Wewak an occasional case only occurred (approximately thirty in each battalion). They were rightly regarded as battalions with excellent antimalarial discipline (following a poor start for which they were only partly responsible). Their arrival at Wewak was, therefore, watched with interest by protagonists of various points of view.

There was a definite increase in case incidence beginning within three to four weeks of their arrival at Wewak. Up to the week ending October 26, 1945, the 30th Australian Infantry Battalion had 20 cases and the 35th Australian Infantry Battalion 48. The highest rate for the 30th Australian Infantry Battalion was nine per thousand per week early in October, whilst the 35th Australian Infantry Battalion suffered 38·6 and 38 per thousand per week in successive weeks in the middle of August. Following this outbreak the members of the latter battalion were ordered to take two tablets daily. There was a subsequent sharp fall to a level of one or two cases a week. These battalions had alternated in resting positions at Alexishafen and forward at Hansa Bay where malaria control depended entirely on the units and patrols were constantly sent into the area near the mouths of the Ramu and Sepik. The maintenance of their low malaria rates throughout a whole year is, therefore, attributable to good antimalarial discipline. There was no operational reason which might lead to gross slackening of this discipline at Wewak, as they took over fixed positions in the last few days of the war and did not engage in offensive operations of any magnitude. If an "Atebrin"-resistant strain was a major factor in the malaria casualties anywhere in the Wewak area it would appear to have been so in this brigade at a late stage of the campaign.

Incidence and Significance of Benign Tertian Malaria.

The suppression of the symptoms of benign tertian malaria has been shown, both experimentally and by experience, to require less "Atebrin" than does malignant tertian malaria. The Land Headquarters Medical Research Unit, Cairns, demonstrated that benign tertian malaria is suppressed by a weekly dosage of 0·4 gramme of "Atebrin". Thus, in operational areas, it is anticipated that benign tertian rates will be low, the disease occurring in that small proportion of troops who, for various reasons, fail to take even four tablets of "Atebrin" a week. Any significant elevation of benign tertian rates must be taken as an index of corresponding failure to take 0·1 gramme daily. This failure is almost certainly more extensive than is indicated by the direct benign tertian : malignant tertian ratio, because, where hyperendemic malignant tertian malaria is present and causes an epidemic in a non-immune population, benign tertian malaria tends to remain latent for the time being at least.

Graph VI presents the malignant tertian and benign tertian cases occurring in the 6th Australian Division. The benign tertian cases paralleled the malignant tertian cases at a lower level. The maximum benign tertian incidence was equivalent to a case rate of 4·6 per thousand per week. In Graph VIII, benign tertian rates from the 6th Aus-



GRAPH VIII.

tralian Division are compared with similar rates from three other operational areas—Solomons, New Britain and Morotai-Borneo. It was pointed out earlier that many troops in all these areas had served in malarious areas before and had contracted benign tertian malaria. It is not possible to say whether this group was proportionately more numerous at Wewak. In any case, it is necessary to explain such a high incidence of this form of the disease in an area where the prescribed minimum dosage of "Atebrin" was 0·7 gramme per week.

The explanation for such a fluctuating high rate of benign tertian malaria must be either that a fair proportion

of the troops were taking less than 0·4 gramme of "Atebrin" per week or that an "Atebrin"-resistant[1] strain or strains of benign tertian malaria were present in the area.

The incidence of mixed benign tertian and malignant tertian infections fluctuated in the same way as the benign tertian and malignant tertian curves.

It is of importance to note that: (i) The benign tertian case incidence during the first peak in which the 19th Australian Infantry Brigade was exclusively involved was proportionately as high as it was in the subsequent main epidemic wave. (ii) It was in this first period that failures in administration were reported from two units under command of the brigade. In the combatant units of the brigade there were, during the six relevant weeks, 29 cases of benign tertian and 185 cases of malignant tertian malaria, a proportion almost identical with that prevailing throughout the whole epidemic period.

The only conclusion possible in terms of present knowledge is that these overt benign tertian attacks must be attributable to defective "Atebrin" intake.

Possibility of an "X" Factor.

Because the consistant experimental results obtained by the Land Headquarters Medical Research Unit at Cairns and the amazingly low rates in Australian troops operating in all other hyperendemic areas had shown the remarkable efficacy of a daily dose of 0·1 gramme of "Atebrin" in suppressing benign tertian malaria and in suppressing and curing malignant tertian malaria, it was generally considered that the "Atebrin" discipline in the division must have fallen below the standard required by General Routine Orders.

On the other hand, doubts regarding the invariable efficacy of "Atebrin" in a dosage of one tablet (0·1 gramme) daily rose in the 6th Australian Division itself, since certain combatant and medical personnel were convinced that malaria casualties were occurring, despite the fact that "Atebrin" was being taken each day with unvarying regularity.

It was suggested that some "X" factor was responsible for the epidemic. In June, 1945, the Director of Medicine was instructed by the Commander-in-Chief, Sir Thomas Blamey, to proceed to Wewak and investigate the problem.

Various factors had to be considered: (*a*) Was "Atebrin" being supplied in the correct dosage and being taken daily with unvarying regularity as laid down in General Routine Orders? (*b*) Was "Atebrin" being absorbed normally and were adequate blood concentrations being built up in troops taking "Atebrin" in the Wewak area? (*c*) Were "Atebrin"-resistant strains of *Plasmodium falciparum* and *Plasmodium vivax* affecting troops in the Aitape-Wewak area, and if so to what extent were they responsible for the epidemic?

The following action was taken immediately: (*a*) A number of specially selected patients—mainly suffering from recurrent malaria contracted in the Aitape-Wewak area—were taken by ambulance plane to Cairns for investigation regarding their utilization of "Atebrin" and in order to isolate local strains in order to investigate possible "Atebrin"-resistance. (*b*) A field section of the Land Headquarters Medical Research Unit was formed, based on Wewak; it included Major I. C. MacDonald as officer in command and Major J. I. Tonge as pathologist, and the appropriate technical staff.

From July to September this field section medical research unit carried out an extensive clinical and parasitological survey amongst troops in the Wewak area, simultaneously collecting specimens of blood for "Atebrin" estimations at Cairns. A special investigation was made at the 104th Casualty Clearing Station of men admitted with overt malaria. During this period hundreds of specimens of blood plasma for "Atebrin" estimations were flown to the Land Headquarters Medical Research Unit at Cairns, as well as some forty selected patients who suffered from overt malaria contracted in the Aitape-Wewak area. (*c*) Lieutenant-Colonel I. M. Mackerras was appointed malariologist, First Australian Army, and proceeded to Wewak to investigate the problem of prevention and control of the epidemic from the entomological aspect.

[1] This possibility has been investigated at Cairns with entirely negative results; no abnormal strain of *Plasmodium vivax* has been detected from Wewak or elsewhere in the South-West Pacific.

The Malaria Hazard in the Wewak Area.

During 1945 the opinion was frequently expressed that malaria infections were more intense in this area than in other parts of New Guinea, the Solomons and New Britain. Existing knowledge regarding "spleen rates" in natives suggested that all these areas would present an equal malaria hazard.

After his appointment as malariologist, First Australian Army, Lieutenant-Colonel I. M. Mackerras investigated this question in July and August, 1945, by determining, firstly, the incidence of malaria infection in local natives, and secondly, the percentage of malaria-infected mosquitoes in the Wewak area. Malaria surveys of natives in villages in the Wewak area showed that infants, children (two to ten years) and adults had a spleen rate of 93%, 90% and 57% respectively, and a parasite rate of 100%, 54% and 2·6% respectively. The species of parasite were *Plasmodium vivax* 51%, *Plasmodium falciparum* 36% and *Plasmodium malariæ* 13%. The carrier rate (gametocytes) was 50% in infants, 6% in children and 2% in adults.

The figures obtained in village children and adults presented lower spleen and parasite rates than in corresponding age groups in Lae obtained by Major T. C. Backhouse. They did show, however, that the area was hyperendemic, and the very real danger of having troops stationed near native villages where children congregate.

It is specially noteworthy that in natives in Wewak the commonest infection at the time of the investigation was benign tertian (51%), not malignant tertian, malaria. When it is remembered that overt malignant tertian malaria was approximately seven times as frequent as overt benign tertian malaria in troops in the Aitape area, it will be realized that a large percentage of troops must have been infected with *Plasmodium vivax* malaria which was being suppressed by "Atebrin".

Entomological surveys of anopheline mosquitoes in villages in the Wewak area showed that in uncontrolled areas 3·5% had oocysts in the stomach and 2·0% sporozoites in the salivary glands (total infected 5·5%). In the controlled areas only 0·2% showed oocysts and 0·2% sporozoites in the salivary gland (total infected 0·4%). It will be seen from these figures that even in uncontrolled village areas only 2% of anopheline mosquitoes (*Anopheles punctulatus*) proved to be infective. In the controlled areas the rate was lower, since many of the older adult mosquitoes had been killed by D.D.T. and tent spraying, and human carriers would be less frequently encountered.

Both native and mosquito surveys lent no support to the view that the risk of infection was higher in the Wewak area than in other hyperendemic areas in New Guinea, the Solomons or New Britain. Apart from temporary variations, the malaria hazard can be regarded as comparable in all these islands. Apart from this the intensity of the malaria infections in volunteers at Cairns vastly exceeded anything that would occur naturally. Volunteers received up to 200 infective bites at Cairns in two months. If the average infective rate of mosquitoes in the jungle is taken at 1%, this would necessitate the individual being bitten by 20,000 anophelines in this period, that is, 333 per night.

Investigations Undertaken by Land Headquarters Medical Research Unit and its Field Detachment.

"Atebrin" Supplied to the Wewak Area.

The "Atebrin" tablets supplied to the 6th Australian Division were similar to those supplied to other parts of New Guinea, the Solomons and New Britain. Notwithstanding, it was felt that investigations should be made to exclude (i) variation in dosage and (ii) wrong processing leading to excessively slow disintegration of the tablets.

Dosage.—Each tablet should contain 0·1 gramme of "Atebrin" hydrochloride. Four different types of tablet in use were analysed by Dr. Adrien Albert, Department of Organic Chemistry at the University of Sydney. The average weight of the "Atebrin" per tablet was as follows: type A, 0·104 gramme; type B, 0·101 gramme; type C, 0·103 gramme; type D, 0·103 gramme. It is evident from these figures that the tablets in all four types contained the required amount of 0·1 gramme or 100 milligrammes per tablet.

Disintegration Times.—In 1942 certain of the "Atebrin" tablets supplied to Australia from the United States of America were found to disintegrate very slowly in water. This was due to four coatings of shellac. The Australian Medical Mission to Washington in 1942 reported this fact to the appropriate authorities in the United States of America, and it was agreed to reduce shellac coating to one layer. Since then disintegration times have been uniformly satisfactory. Three of these four types of tablets tested disintegrated rapidly. In type D the time taken was found to vary from five to fifteen minutes, but in no instance would this have been sufficient to lead to defective disintegration after the tablet was swallowed with resultant defective absorption.

The only observation of interest derived from this particular investigation was the fact that certain shellac-coated tablets (one coat) could be retained in the mouth throughout breakfast or morning tea and subsequently removed from the mouth intact. This would afford opportunity for circumventing effective "Atebrin" administration, even when the "Atebrin" was given on parade, should an individual decide to do so.

"Atebrin" Plasma Concentrations.

Another "X" factor suggested to explain the malaria casualties was that though "Atebrin" was taken regularly, it was not being effectively absorbed under the arduous conditions of the Aitape campaign, or if it was absorbed, that the drug was being abnormally rapidly degraded or excreted. Irregular taking of "Atebrin" or any of these other factors would result in low "Atebrin" blood levels and minimal tissue storage and would permit parasites to increase in the circulatory blood until they attained a density necessary to produce an overt attack.

Specially Selected Patients from Wewak Investigated at Cairns.—In the earliest stage of the investigation serial plasma "Atebrin" estimations were made on a series of patients flown from Wewak to Cairns. The initial cases were chosen because of repeated attacks following standard quinine, "Atebrin" and "Plasmoquine" ("Q.A.P.") treatment. The first three were of considerable interest, and for this reason will be recorded in detail.

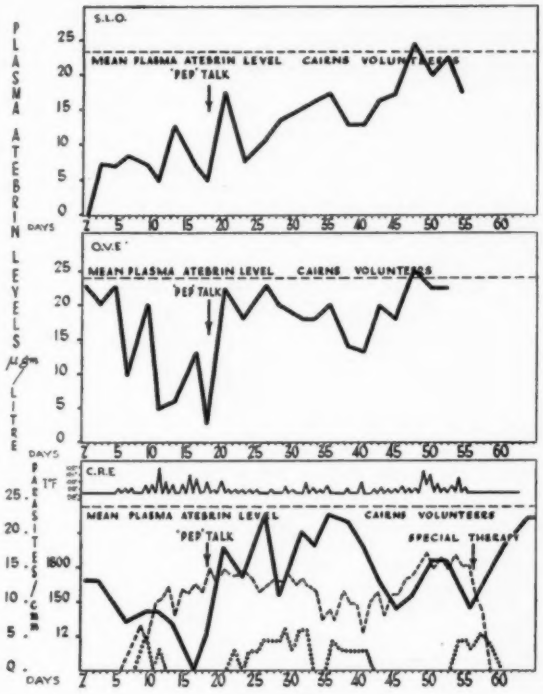
CASE I.—S.L.O., a private in an infantry battalion, aged twenty-eight years, served in the Aitape-Wewak area from December, 1944, to June, 1945, and stated that he took one tablet of "Atebrin" daily. His previous malaria history was that in previous campaigns he had had two overt attacks of *Plasmodium vivax* malaria in late 1942, three during 1943, and a clinical attack (unconfirmed) in March, 1944. His present malaria history was that in the Aitape-Wewak area he had a clinical attack (unconfirmed) in April, 1945, and three attacks of *Plasmodium falciparum* malaria on April 16, May 3 and May 21, 1945, respectively.

The outstanding feature of the history of this patient was the rapidity with which he was having recrudescences of malignant tertian malaria. He was sent from Aitape to the 2/7 General Hospital, Lae, and there it was noted that the urinary "Atebrin" levels were low, despite the fact that he was receiving 0·1 gramme of "Atebrin" daily either in solid or liquid form under supervision. Subsequently he was flown to the Land Headquarters Malaria Research Unit for special investigation at Cairns.

On admission, the patient had moderate "Atebrin" staining of the skin; the spleen was palpable three fingers' breadth below the left costal margin. The plasma "Atebrin" levels equalled 6·0 to 8·0 micrograms per litre.

He was placed on a regimen of 100 milligrammes of "Atebrin" daily under supervision. Plasma "Atebrin" levels gradually rose to satisfactory levels (see Graph IX). No parasites were even demonstrated in thick films during the ninety days he was under observation there. He gained

SOLDIERS SENT TO CAIRNS FROM AITAPE, WEWAK AREAS TAKING ONE TABLET OF ATEBRIN DAILY



GRAPH IX.

fifteen pounds in weight, became physically fit and was subsequently sent south.

CASE II.—O.V.E., a lance-corporal in an infantry battalion, aged twenty-three years, served in Aitape-Wewak from December, 1944, to June, 1945. He stated that he took one tablet of "Atebrin" daily.

Previous attacks of *Plasmodium falciparum* malaria had occurred on April 17, May 21 and June 3, 1945. He was admitted to Cairns on June 16, 1945, two days after the end of a standard "Q.A.P." course of treatment for malignant tertian malaria. There was moderate "Atebrin" staining of the skin; the spleen was palpable to one finger's breadth below the left costal margin; the liver was palpable and slightly tender.

This soldier was given 100 milligrammes of "Atebrin" daily. Though his plasma "Atebrin" level on admission was satisfactory, it fell steadily during the next two weeks at Cairns until the low level of two microgrammes per litre was reached on the fifteenth day (see Graph IX). He was full of complaints, was an unstable nervous type, and finally was detected chewing gum on the "Atebrin" parade. The latter is a well-known dodge for avoiding the swallowing of the "Atebrin" tablet. At this stage he was interrogated and was informed that there was little chance of his getting leave south as long as his attacks of malaria continued. Subsequently there was a very rapid rise in the "Atebrin" plasma levels and these were well maintained over a period of 75 days' observation. Blood films were "negative" for parasites throughout this period and clinical malaria did not develop.

CASE III.—C.R.E., a corporal in an infantry battalion, aged twenty-two years, served in Aitape-Wewak. He stated that he took one tablet of "Atebrin" daily. He suffered from attacks of *Plasmodium falciparum* malaria on April 1 and May 1, 1945, and from attacks of *Plasmodium vivax* on May 13 and June 5, 1945.

Admitted to Cairns after the last dose of therapy for an attack of malaria on June 5, 1945, he was put on a regimen of "Atebrin" of 100 milligrammes daily. On June 22, 1945, seven days after his admission, *Plasmodium falciparum* trophozoites were demonstrated in thick blood films. By

June 28, 1945, densities of 364 per cubic millimetre were present and the plasma "Atebrin" concentration fell to 0·0 microgramme per litre. This man was also informed that there was little prospect of his going south on leave while malaria attacks continued. Three days later the "Atebrin" levels rapidly rose and subsequently consistently remained over ten microgrammes per litre, and on occasions reached twenty microgrammes per litre. In spite of these levels (see Graph IX) *Plasmodium falciparum* trophozoites persisted until August 11, 1945, when 100 milligrammes of "Paludrine" were administered; forty-eight hours later blood films were "negative" for *Plasmodium falciparum* trophozoites and remained so for fifteen days when the blood smears again became positive, in spite of the continued administration of "Atebrin" 100 milligrammes daily. On August 31, 1945, administration of "Atebrin" 200 milligrammes daily was commenced, but failed to control the symptoms or to render blood films "negative", so the administration of "Paludrine" 300 milligrammes daily for ten days was commenced on September 11, 1945. This soldier was seldom confined to bed; he did not develop a satisfactory gametocyte wave (maximum density was 16 per cubic millimetre), and maintained plasma "Atebrin" concentrations below the mean expected on a regimen of 100 milligrammes daily. His plasma "Atebrin" level two days after his admission was 12 microgrammes per litre, which was very low, as it was stated that he had finished his routine malaria treatment on the day of admission; this course consisted of 2·7 grammes of "Atebrin" administered over a period of eight days.

In the first case, that of S.L.O., overt malaria failed to develop over a three-months period of observation at Cairns; the plasma "Atebrin" levels rose steadily and there was no evidence that "Atebrin" was not being absorbed or that it was being degraded abnormally. The recurrent attacks at Wewak and Lae, and the poor excretion in the urine, are explicable on the basis that by one means or another "Atebrin" was avoided in New Guinea.

In the second and the third cases there was abundant evidence that both soldiers had been evading "Atebrin" at Cairns. The dramatic rise in plasma "Atebrin" concentrations which followed their interrogation could have occurred only if several "Atebrin" tablets had been taken in the intervening two or three days. The increasing density of malignant tertian parasites in the blood of the third soldier (C.R.E.), despite reasonable "Atebrin" concentrations, constituted the first evidence that we might be dealing with an "Atebrin"-resistant strain. If so, it was appearing in an individual who had obviously been taking "Atebrin" in sub-optimal dosage, and it suggested that in his case at least poor "Atebrin" discipline might have been the underlying cause.

TABLE XVI.

Mean Plasma-"Atebrin" Levels in Men Stated to be Taking 0·7 Gramme Weekly.

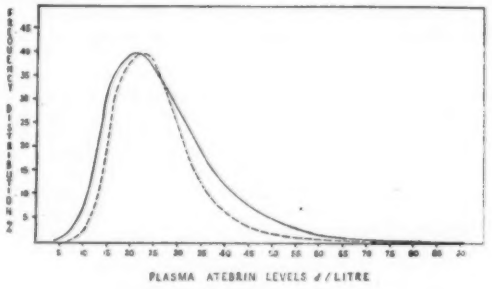
Category.	Number Estimated.	Mean. (Microgrammes per Litre.)
1. No past history of malaria, no symptoms, no parasites	333	21·14
2. Past malaria in campaign. No symptoms, no parasites	25	19·45
3. No past history. Symptoms present. No parasites	83	21·78
4. Past history of malaria. Symptoms present, no parasites	16	19·52
5. Parasites found in blood films	20	21·13
6. Patients admitted with overt malaria	42	27·57
Total (0·7 gramme weekly)	519	21·56
Total (0·7 gramme weekly less patients)	477	21·11
0·7 gramme weekly with some evidence of past or present infection	144	21·02

Investigation of Troops while Operating in the Wewak Area.—Plasma specimens were collected by the field section research unit according to standard methods and forwarded by plane to the Land Headquarters Medical Research Unit, Cairns, where fluorometric determinations were made by means of the double extraction method of Brodie and Undenfriend.

In the presentation of results the following procedures were adopted: (i) Primary classification of samples was made according to the dosage which the soldier stated he was taking at the time of examination. (ii) Subsequent classification of these main groups was made as indicated in the tables. (iii) Readings were recorded only in those cases in which the last tablet was taken not less than twenty-one hours or more than twenty-seven hours before the plasma specimen was taken. (iv) All means were calculated geometrically. (v) Readings were expressed in microgrammes per litre.

The results obtained will now be considered.

A comparison with the results obtained in Cairns volunteers shows a close agreement between mean levels and standard deviations in the two series (Graph X and Table XVII).



GRAPH X.

Showing frequency distribution of plasma "Atebrin" levels—soldiers in 6th Australian Division stated to be having 0·7 gramme of "Atebrin" weekly. The continuous line represents observations made at Cairns; the interrupted line represents observations made at Aitape-Wewak.

TABLE XVII.

Group.	Mean. (Microgrammes per Litre.)	Standard Deviation.
Aitape-Wewak	21·56	1·37
Cairns	20·12	1·59

The only members of the Aitape-Wewak group with anomalous levels were patients admitted with overt malaria. Their mean level of 27·57 microgrammes per litre was significantly high. The only likely explanation of this finding is that soldiers developing an attack would not unnaturally have taken extra "Atebrin" on the onset of suspicious symptoms, thus producing temporarily higher levels.

These recorded levels are in accord with the statements made by the men, that is, that they took extra tablets occasionally, perhaps once or twice weekly, to be "on the safe side".

TABLE XVIII.

Mean Plasma-"Atebrin" Concentrations in Men Stated to be Taking 0·7 Gramme Weekly plus an Occasional Extra Tablet.

Category.	Number Observed.	Mean. (Microgrammes per Litre.)
No past malaria, no symptoms, no parasites	17	26·54
All members of group	53	24·85

The most important feature of these groups is that none of the mean levels approaches the level obtained at Cairns in volunteers taking this dosage.

Even those men at Wewak who had recently been given a full antimalarial course and subsequently took a maintenance dose of 0·2 gramme daily did not develop plasma concentrations comparable with those at Cairns. The Cairns figures on volunteers provide a reliable standard because of the rigidly controlled conditions under which the unit works; in addition the results are in agreement with those of American workers with extensive experience in plasma "Atebrin" technique.

TABLE XIX.

Mean Plasma-"Atebrin" Concentrations in Men Stated to be Taking 1·4 Grammes Weekly.

Category.	Number Observed.	Mean. (Microgrammes per Litre.)
1. No past malaria, no symptoms, no parasites	86	30·88
2. Past malaria in campaigns, no symptoms, no parasites	24	33·07
3. Patients with overt malaria	26	32·48
4. Maintenance after therapy	60	29·24
Whole group	155	31·70
Whole group less patients	129	31·55

TABLE XX.

Group.	Number Observed.	Mean. (Microgrammes per Litre.)
Aitape-Wewak	155	31·70
Cairns	124	43·1

Conclusions drawn from the tabulated data based on analysis of specimens collected at Wewak are as follows:

1. Plasma "Atebrin" levels in men of the 6th Australian Division stated to be taking 0·7 gramme weekly were satisfactory in that the mean "Atebrin" levels were 21 microgrammes per litre or more. No evidence was obtained in this group that there was any anomaly in the absorption, degradation or excretion of this drug in troops in Wewak compared with volunteers in Cairns.

2. Anomalous features were noted in patients admitted to hospital with malaria and in soldiers stated to be taking 1·4 grammes of "Atebrin" weekly. The only overall explanation which might cover all the facts observed is that irregularities existed in "Atebrin" intake.

3. It is considered that the plasma "Atebrin" levels found in the 6th Australian Division indicated that "Atebrin" consumption was, in general, adequate to confer protection against all strains of malignant tertian malaria previously worked with at Cairns. This conclusion naturally applies only to the period in which the samples were taken, that is, in July and August towards the end of the epidemic.

Spleen and Parasite Rates in Troops in the 6th Australian Division.

A survey was also made to determine the degree of malaria in ambulatory men of the 6th Australian Division. In Table XXI a comparison is made of the results obtained with those from other areas.

It is evident that towards the end of the Wewak epidemic clinical and parasitological findings in men walking about in apparent good health were similar to those found in earlier campaigns when virtually every man was infected. Bougainville and Morotai-Borneo present a completely different picture, indicating very adequate suppression of malaria.

Judging from the experience of the earlier campaigns, following which repeated and widespread relapses of malaria occurred, it is reasonable to estimate that no less

TABLE XXI.

Comparison of Spleen and Parasite Rates in Soldiers Operating in Different Campaigns.

Group.	Number Examined.	Spleen Rate *per Centum.*	Parasite Rate *per Centum.*	Gametocytes *per Centum.*
Wewak—				
1. May, 1945	225	—	5·0	2·0
2. July, 1945	423	11·0	5·5	2·5
Wewak, Prince Alexander Mountains, August, 1945	113	—	1·0	1·0
Milne Bay, 1943 (Mackerras)	295	7·0	12·0	7·0
Buna-Gona, 1943 (Ferris)	903	4·0	16·0	11·0
Bougainville, June, 1945 (Sewell)	300	0·0	1·0	1·0
Morotai, Labuan and Balikpapan (July-August, 1945)	1,740	Not recorded	0·17	0·05

than 90% of the force at Wewak was infected at some time during the period under review. It would therefore appear that "Atebrin" had effectively suppressed at least two out of every three malignant tertian malaria infections and a much larger proportion of benign tertian infections. If this is so, it is evident that the majority of troops infected with malaria at Wewak could not have been infected by an "Atebrin"-resistant strain of parasite.

Early in the investigation malaria parasites were found in the blood of individuals who were either quite well or had such slight symptoms that they continued to work efficiently. Seven out of 65 men who were kept under observation for six weeks (the officer in command of the detachment administered their "Atebrin" daily) had transient parasitæmia including the appearance of gametocytes. In one case demonstrable parasites persisted for a month without the development of an overt attack. This individual had as many as 3,000 trophozoites of *Plasmodium falciparum* per cubic millimetre. Sixty days after the termination of the investigation he was still well, no symptoms having developed. Only one of the seven was admitted to hospital with a mild overt attack. Parasites, fever and symptoms disappeared on the second day of routine treatment.

Amongst another group of ninety men, seven were found, on initial examination, to have trophozoites of *Plasmodium falciparum* in their blood. These men were kept under observation for two and a half weeks, during which time four observations were made on each man. Fluctuating parasitæmia with gametocyte formation was detected. A month later one of them was admitted to hospital with an overt attack of malaria.

Thus 14 of 155 men (9%) were shown to have malaria parasites demonstrable in thick blood films without symptoms. Two only subsequently developed overt attacks of malaria. In all, 38 plasma "Atebrin" estimations were made on these fourteen individuals. A mean level of 23·66 microgrammes per litre was found. Two of them were stated to be taking 0·2 gramme daily for a part of this period of investigation.

Only three had suffered previous malaria attacks, two of them a few weeks before being first examined, one six months before. The histories of the former were:

No. 439 ("Atebrin" administered by the officer commanding the field detachment). The patient had malignant tertian malaria one month prior to the first examination. He was taking two tablets daily. On the first examination no parasites were found. The plasma contained 38 microgrammes of "Atebrin" per litre. A fortnight later there were five trophozoites per cubic millimetre. The plasma "Atebrin" content was 32 microgrammes per litre. After a month, when he had resumed taking one tablet of "Atebrin" daily, there were 160 trophozoites and 10 gametocytes per cubic millimetre. The plasma "Atebrin" content was 24·0 microgrammes per litre. Six weeks later there were 60 gametocytes per cubic millimetre. The plasma "Atebrin"

content was 21 microgrammes per litre. No symptoms were present thoughout.

No. 496. The patient suffered from malignant tertian malaria on June 17, 1945. He was examined on August 4, 1945. He was taking two tablets daily as a follow-up course. Blood smears showed ten trophozoites per cubic millimetre. The plasma "Atebrin" content was 30 microgrammes per litre. On August 14 there were 360 trophozoites per cubic millimetre, and on August 19, 720 trophozoites per cubic millimetre. The plasma "Atebrin" content was 49 microgrammes per litre.

The presence of parasites, *Plasmodium falciparum*, in people with a theoretically effective plasma "Atebrin" concentration had not previously been shown to occur except in one of the Wewak cases from Cairns. This evidence supported the possibility that a strain or strains of *Plasmodium falciparum* relatively resistant to "Atebrin" might exist in this area.

Nature of Overt Malaria at Wewak.

Observations were made on the severity of clinical attacks of malaria, which in general were remarkably mild. In a series of 100 patients during standard courses of treatment demonstrable parasites persisted for an average period of 2·2 days, whilst fever lasted only 2·3 days. Forty-five cerebral infections were reported up to the end of August with four deaths. An examination of the history sheets of 35 of these men has been made. Only 24 could be regarded as certain sufferers from cerebral malaria. The others all had "negative" blood films and clinical features which were not necessarily diagnostic of malaria.

Considering the size of the epidemic, the low incidence of severe illness due to malignant tertian malaria was striking. "Atebrin" modified the clinical picture and severity of the disease even when breakdown occurred and overt malaria became manifest.

The field detachment research unit was thus able to show that: (i) Late in the epidemic satisfactory quantities of "Atebrin" were being taken by the 6th Australian Division as a whole. (ii) The degree of infection in the division exceeded the reported malaria rate. (iii) A proportion of apparently healthy men had demonstrable parasites whilst taking the prescribed amount of "Atebrin", that is, one tablet daily. (iv) The severity of the clinical attacks were markedly diminished by the "Atebrin" taken.

Relative "Atebrin" Resistance of Strains of Plasmodium Falciparum and Plasmodium Vivax.

Plasmodium Falciparum.—The investigations at Cairns were designed primarily to determine if there were in soldiers serving in the Aitape-Wewak area strains of *Plasmodium falciparum* that were relatively "Atebrin"-resistant.

Soldiers who were considered most likely to be infected with relatively "Atebrin"-resistant strains were selected and sent by air to Cairns. It was considered that those soldiers who had had repeated attacks of malignant tertian malaria in a short period of time (that is, those most likely to have had recrudescences while receiving "Atebrin" maintenance after concluding a standard course of therapy), those with cerebral malaria, and those with hyperinfections were most suitable for the investigation.

Nine "strains" of *Plasmodium falciparum* were isolated from nine soldiers sent to Cairns and mosquitoes were infected. Subsequently volunteers receiving "Atebrin" or other antimalaria drugs in suppressive dosage were bitten by these mosquitoes; the volunteers were infected with one or more of these nine "strains".

These experiments showed that seven of the nine "strains" isolated from these specially selected soldiers were atypical in that malignant tertian malaria was not suppressed, or if suppressed not cured, in volunteers taking 100 milligrammes of "Atebrin" daily. Furthermore, malignant tertian malaria was sometimes suppressed, but not readily cured, in volunteers receiving 200 milligrammes of "Atebrin" daily. Both of these suppressive regimens were continued for twenty-eight days after the last exposure to the bites of infective mosquitoes.

The "strains" of *Plasmodium falciparum* isolated from two of these soldiers behaved normally in volunteers receiving 100 milligrammes of "Atebrin" daily, in that the infection was competely suppressed and radically cured when the daily dose of "Atebrin" was continued over the period of biting and subsequent twenty-eight days. The donors of these two "strains" were both experiencing their first attacks of malaria.

Of four volunteers developing malignant malaria, who were treated with the standard course of treatment employed at Aitape-Wewak at the time, three had recrudescences while taking 100 milligrammes of "Atebrin" daily as maintenance. This result was most unusual, and its significance in regard to the prevalence of this "Atebrin"-resistant strain was considerable. Since the incidence of secondary attacks in the Aitape-Wewak campaign was only 1·15 to 1·3, it follows that failure to establish radical cure was exceptional, and that the common strain producing malaria there was "Atebrin" sensitive and not "Atebrin" resistant.

The greater the daily dose of "Atebrin" and the higher the mean plasma "Atebrin" concentration, the greater the degree of suppression obtained in volunteers infected with relatively "Atebrin"-resistant strains of *Plasmodium falciparum*.

Evidence of altered sensitivity to "Santochin" was also obtained in two of the four volunteers receiving 100 milligrammes of this drug each day. These two volunteers showed full suppression, but were not cured of their infection, a result not previously observed at Cairns. No alteration in sensitivity to quinine, "Resochin", sulphadiazine or "Paludrine" was observed.

Possible evidence of loss of "Atebrin" resistance was obtained in one strain after the fourth passage through mosquitoes and man, full suppression and radical cure was obtained in a volunteer receiving 100 milligrammes of "Atebrin" daily who received ten infected bites with a strain hitherto regarded as particularly resistant to "Atebrin".

Plasmodium Vivax.—A single strain of *Plasmodium vivax* isolated from a soldier infected in the Aitape-Wewak area was tested in volunteers receiving either 700 or 400 milligrammes of "Atebrin" each week. The infections in volunteers receiving both regimens were fully suppressed, but the volunteers developed typical benign tertian malaria three to five weeks after ceasing to take "Atebrin". No evidence of altered sensitivity to "Atebrin" was obtained with this strain of *Plasmodium vivax*.

Summary.—These findings may be summarized as follows.

1. Two main groups of "strains" of *Plasmodium falciparum*, with regard to "Atebrin" sensitivity, were found: (*a*) strains that were irregularly suppressed, but not destroyed, by 100 milligrammes of "Atebrin" daily in volunteers exposed to experimental mosquito transmitted infection; in volunteers receiving 200 milligrammes of "Atebrin" daily full suppression was sometimes achieved, and they were radically cured of their infection with these strains. (*b*) Strains which were normally sensitive to "Atebrin" and which were fully suppressed and destroyed in volunteers receiving 100 milligrammes of "Atebrin" daily. This group conformed in behaviour to all other strains of *Plasmodium falciparum* investigated at Cairns.

2. No relatively "Atebrin"-resistant strain of *Plasmodium vivax* was encountered. One strain investigated was normally sensitive to "Atebrin" and was fully suppressed in volunteers receiving 400 milligrammes of "Atebrin" weekly.

Summary.

In the Aitape-Wewak campaign some 27·6% of the 6th Australian Division and 9·8% of the troops in the 3rd Australian Base Sub-Area suffered from one or more overt attacks of malaria in a period of approximately eleven months. There is reason to believe that during this period of exposure at least 90% of the troops would have been infected with *Plasmodium falciparum* or *Plasmodium vivax* or both. If so, then "Atebrin" was effective in suppressing overt attacks of malaria in 66·7% of the total force who were infected.

During the campaign the incidence of overt malaria attacks in officers was approximately one-half that among non-commissioned officers and men.

Malignant tertian malaria predominated, the type of malaria being *Plasmodium falciparum* in 78·2% of the total cases and *Plasmodium vivax* in 12·8%. In 11·1% of individuals, the clinical diagnosis was not confirmed by demonstration of parasites in blood smears.

The proportion of men suffering from multiple attacks was not high, the average attack per patient being 1·3 in troops of the 6th Australian Division and 1·15 in the 3rd Australian Base Sub-Area troops.

The initial rise in malaria casualties developed in two units of the 19th Australian Infantry Brigade in January-February, 1945. Investigation revealed administrative failure in these two units and disciplinary action was taken. The highest rates were reached in the 16th Australian Infantry Brigade (113 per 1,000) in the week ending May 4 and 110 per 1,000 per week in the week ending June 8. In the latter week the 19th Australian Infantry Brigade reached a maximum rate of 65 per 1,000 per week. The 17th Australian Infantry Brigade reached a rate of 20 per 1,000 per week in the week ending April 20 and 30 per 1,000 per week in the week ending July 6. By September, one month after the end of the war, rates had fallen to a relatively low level.

At times during this period the "Atebrin" regimen was increased from one to two tablets daily with variable results in the different brigades.

Though malignant tertian infections largely predominated, the incidence of *Plasmodium vivax* infections followed the *Plasmodium falciparum* cases at a lower level. In all there were 741 attacks of overt benign tertian malaria in the Aitape-Wewak area; the maximum benign tertian case rate was 4·6 per 1,000 per week.

The possibility that some peculiar "X" factor was responsible for the epidemic was investigated by the Land Headquarters Medical Research Unit and its field detachment, some forty specially selected malaria patients and hundreds of specimens of plasma for "Atebrin" estimations being flown to Cairns by ambulance aeroplane for the purpose.

The average weight of the "Atebrin" per tablet being used in the Aitape-Wewak area was proved to be correct (0·1 gramme) and the disintegration times fell within normal limits.

The plasma "Atebrin" levels in troops stated to be taking 100 milligrammes of "Atebrin" daily did not significantly differ from those obtained in volunteers receiving the same daily dosage at Cairns—the Aitape-Wewak concentrations had a $mean_G$ of 21·56 microgrammes per litre (519 observations) with a standard deviation of 1·37, whereas the Cairns $mean_G$ was 21·00 microgrammes per litre (790 observations) with a standard deviation of 1·49. However, the plasma "Atebrin" concentrations in soldiers stated to be receiving 200 milligrammes of "Atebrin" daily were considerably lower than concentrations observed in volunteers receiving similar dosage at Cairns—the concentrations were: Aitape-Wewak $mean_G$ 31·7 microgrammes per litre (155 readings), Cairns 43·3 microgrammes per litre (147 readings).

A proportion of apparently healthy men were found to have demonstrable parasites and/or palpable spleens whilst taking the prescribed amount of "Atebrin", that is, one tablet daily. In the majority of instances parasites later disappeared without producing overt attacks of malaria. It is evident from these observations that the degree of infection in the division exceeded the reported malaria rate, since such individuals were not admitted to hospital for treatment.

Investigations at Cairns were carried out to determine if there were strains of *Plasmodium falciparum* in soldiers serving in the Aitape-Wewak area that were relatively "Atebrin" resistant. Two main strains of *Plasmodium falciparum* were found: (*a*) Strains that were irregularly suppressed but not radically cured by 100 milligrammes of "Atebrin" daily in volunteers exposed to soporozoite induced infection. Sometimes 200 milligrammes fully suppressed and radically cured this infection. (*b*) Strains which were normally sensitive to "Atebrin" and which were fully suppressed and eliminated, provided the volunteers were receiving 100 milligrammes of "Atebrin" daily.

Another characteristic of this relatively "Atebrin"-resistant strain of *Plasmodium falciparum* was the failure of routine quinine, "Atebrin" and "Plasmoquine" treatment to produce radical cure. Such a result was most unusual and had been only rarely observed previously. Its significance in regard to the prevalence of this strain throughout the Aitape-Wewak campaign was considerable. Since the incidence of secondary attacks in treated malaria patients was 1·15 for troops in the 3rd Australian Base Sub-Area and 1·3 for troops in the 6th Australian Division, it follows that failure to establish radical cure was exceptional in the area and that the prevalent strain producing overt malaria there was the usual "Atebrin"-sensitive and not the relatively "Atebrin"-resistant type.

The origin of the Aitape-Wewak relatively "Atebrin"-resistant strain or strains is a matter of conjecture. The fact that it was first found in an individual who had purposely been avoiding taking "Atebrin", suggested that it might be arising like strains of bacteria resistant to sulphonamides or strains of trypanosomes resistant to arsenic, as a result of sub-optimal dosage rather than that it constituted an inherent biological characteristic of a strain of *Plasmodium falciparum* limited to the Aitape-Wewak coastline of New Guinea. Certainly the strain could not be described as being "Atebrin"-fast in the ordinary acceptance of that term. Many transitions were noted from individuals on one tablet daily who showed microscopic densities of parasites (*Plasmodium falciparum*) in the blood without developing overt attacks, to those in whom a regimen of two tablets daily not only failed to produce a radical cure, but also failed to suppress overt attacks. These observations suggested that the quality of "Atebrin" resistance was in process of production. Possible evidence of loss of "Atebrin" resistance in one experiment raised the question of the reversion of the strain to one of normal "Atebrin" sensitivity after the fourth passage through man. Time, unfortunately, did not permit full investigation of this interesting possibility.

This relatively "Atebrin"-resistant strain was suppressed effectively by "Santochin" in a dosage of one tablet daily (0·1 gramme), but not necessarily eliminated. On the other hand, it manifested no increased resistance to "Resochin", "Paludrine", quinine or sulphadiazine, the strain behaving in all respects normally regarding these drugs.

No experimental or clinical evidence of a relatively "Atebrin"-resistant strain of *Plasmodium vivax* was encountered in patients from Wewak. The strain of *Plasmodium vivax* from Wewak studied at Cairns behaved normally in all respects and was equally well suppressed in volunteers, whether receiving four tablets (0·4 gramme) or seven tablets (0·7 gramme) weekly. It follows that the 741 attacks of overt benign tertian malaria developing in troops in the Aitape-Wewak area must be attributable to a failure to take even four tablets weekly.

Conclusions.

1. It is estimated that at least 90% of troops operating in the hyperendemic Aitape-Wewak area were infected with *Plasmodium falciparum* or *Plasmodium vivax*, or both parasites. Only 23·3% of the whole force developed overt malaria. It follows that "Atebrin" was effective in suppressing major clinical symptoms in 66·7% of members of the force.

2. Extensive investigations indicated that of the overt malaria attacks contracted by 23·3% of the force, the underlying cause was defective "Atebrin" discipline in all cases of *Plasmodium vivax* malaria and in over two-thirds of the cases of *Plasmodium falciparum* malaria.

3. An undetermined proportion of one-third of the cases of overt malignant tertian malaria are explicable on the basis of infection with a relatively "Atebrin"-

resistant strain of *Plasmodium falciparum*. In troops infected with such a strain, one tablet (0·1 gramme) of "Atebrin", even though taken every day with absolute regularity, would not be effective in suppressing malaria. Two tablets daily would generally suppress overt attacks, but not necessarily produce radical cure.

4. Plasma "Atebrin" estimations made in July and August revealed that while "Atebrin" was being purposely avoided by some exceptional individuals, the dosage being taken by the 6th Australian Division as a whole was adequate—the plasma "Atebrin" levels in troops at Wewak stated to be receiving 100 milligrammes of "Atebrin" daily equalling those attained in volunteers at Cairns in the same dosage regimen.

5. The factor or factors responsible for the production of a relatively resistant strain or strains of *Plasmodium falciparum* in the Wewak area were not finally determined. The strain was certainly not "Atebrin"-fast, and numerous observations suggested that the quality of "Atebrin" resistance was one "in the making", rather than a fixed biological characteristic of a strain of *Plasmodium falciparum* peculiar to the Aitape-Wewak area. If so, sub-optimal dosage due to a failure to take the prescribed dosage of "Atebrin" daily may have been the responsible factor.

No increased resistance was manifest by the relatively "Atebrin"-resistant Wewak strain of *Plasmodium falciparum* to antimalaria drugs, other than "Atebrin" and "Santochin". If available, the substitution of "Paludrine" or "Resochin" in appropriate dosage would have effectively suppressed and cured malaria in troops infected with the relatively "Atebrin"-resistant Wewak strain (*Plasmodium falciparum*).

DEFICIENCY DISEASES.[1]

By B. L. W. CLARKE,
Brisbane.

IT is with a certain amount of trepidation that I address you this evening, because I feel that there are others who know much more about this subject.

When I was commissioned in the Australian Imperial Force in 1941, it was as a radiologist and not as a physician. I must admit that at that time I took little or no interest in dietetics or vitamin deficiency. I did, however, have some idea that scurvy was due to the absence of vitamin *C*, and that X-ray films frequently revealed subperiosteal hæmorrhages in children who had suffered from scurvy; but I do not remember having heard of a case of scurvy amongst our men in Malaya. However, I shall endeavour to give you a picture of some of the medical conditions encountered while we were guests of the Imperial Japanese Army.

Much of what I say will lack academic confirmation, for the simple reason that we had not the material to carry out research work, though we had in the camp many eminent physicians and scientists who gave valuable advice. Our "hosts" in the early stages removed about one-third of our medical equipment, and during the next three and a half years made further inroads on our lessening supplies without ever attempting to replace anything. Even as late as February, 1945, they removed a number of our remaining microscopes. I venture to state at the very outset that over 75% of our afflictions would never have occurred if we had had access to the elementary amenities of civilization and had been allowed to exercise hygiene control and carry out the basic principles of preventive medicine. In this respect the most outstanding feature was that Singapore and in fact a large area of Malaya was free from malaria prior to the entry of the Japanese, and that during their occupancy the whole system of anti-malaria control broke down and no attempt was made by them to remedy it, with the result that about 90% of the prisoners of war contracted one or other form of malaria. The Japanese contracted malaria as well.

In the first few months of our captivity the water supply had failed, and we were strictly limited to a small quantity of water, even as little as one quart per day per man for all purposes. This water was, of course, contaminated and had to be treated before it could be consumed. After the first six months we had a good and plentiful water supply in Changi.

In the early stages we lacked much gear, and disposal of refuse could not be carried out efficiently, so that in a short time flies appeared by the millions and dysentery broke out and affected the majority of our personnel. Dysentery remained with us right up to the end, as fresh patients were always coming in from other areas.

We were afflicted with many lesions about which we had never previously heard, and on reference to our limited literature frequently only a passing note or no mention at all was found.

In the internment camp the men were crowded together; the actual space for one man was six feet by three feet, so within the hospital wards the beds were so tightly packed together that it was almost impossible to pass between them, and in the same area we had to house all messing and hospital gear.

The rations in our possession when we were captured lasted about ten days, and then our "hosts" announced our future daily ration, as follows: rice (polished), 500 grammes; flour, 50 grammes; meat or fish, 50 grammes; vegetables, 100 grammes; milk, 15 grammes; sugar, 20 grammes; ghie, 5·0 grammes; and ten cigarettes per week. (Note the absence of salt.) Of this ration we received the 500 grammes of rice per day and a meat issue of 100 grammes per week. They forgot the rest. The meat supply stopped in about five months, and then no more came in for seventeen months, when a small issue was given for a few weeks.

In the first two years they sent us some fish, dried and fresh. "Fresh" fish was usually unfit for human consumption, and was used either to fertilize our own gardens or to feed our own ducks. In later years when the Americans bombed the shipping in Singapore, the fish ration increased considerably; many sharks, stingrays and other types were killed in the bombing and floated ashore.

It was pointed out to the Japanese that this diet was deficient in vitamins, protein and fat. We predicted that within eight weeks beriberi would be the first of the deficiency diseases to affect us. However, they were not interested. No vegetables arrived for approximately four months. At the end of May, 1942, we had an issue of one banana between three men. The vegetable, which consisted of greens somewhat like silver beet (it has a variety of local names) and green papaws, arrived in reasonable quantities about June, 1942. In the meantime we had established our own gardens.

Within a few days of going on this coolie diet the majority of the troops became constipated. One or two bowel actions per week was the average. On the other hand, enormous quantities of urine were passed. Day and night urination was about equal. Four to five times per night was not uncommon, large quantities being passed each time. Curiously enough we never became thirsty. Œdema of the ankles appeared in about four weeks' time. This œdema was usually mild and disappeared with rest, to recur while the subject was walking about.

Beriberi.

Beriberi commenced in April, 1942. I shall here make only a few brief remarks about this disease, as the subject is well covered in textbooks such as that of Manson-Bahr. I think I am safe in saying that there are four distinct varieties, as follows.

1. *Neuritic Beriberi.*—In the neuritic form wasting of the muscles of the legs and arms occurred. The skin became dried and somewhat lichenified and anæsthesia developed. Sometimes the abdominal wall was involved. At the same time various groups of muscles became

[1] Read at a meeting of the Queensland Branch of the British Medical Association on April 5, 1946.

paralysed. Foot drop and wrist drop were the most common, but often whole limbs were involved, and I have seen the condition involve other areas such as the lip, tongue, palate and orbital muscles. In the majority of cases paralysis was symmetrical. Operations were possible without anæsthesia.

2. *Beriberi Œdema.*—The earliest symptom was œdema commencing in the most dependent parts of the body, usually the feet, and gradually spreading up the legs, across the abdominal wall and as far as the face. Œdema of the scrotum was one of the most disturbing symptoms, reaching tremendous proportions. In fact, the amount of œdema was limited only by the capacity of the skin to remain intact. Skin rupture was not uncommon in later years. We had to distinguish between famine œdema and beriberi œdema, and mild infections were often rapidly followed by œdema.

3. *Cardiac Beriberi.*—I think cardiac beriberi was responsible for the sudden death of many men who had no apparent complaint. The heart became greatly enlarged, the right side in particular being involved. The blood pressure fell very low, and systolic pressures of 90 millimetres of mercury and diastolic pressures of 50 to 60 millimetres of mercury were not uncommon. Through the stethoscope the heart sounds were heard to be evenly spaced and reminded one of a pendulum swing. There was also the usual train of symptoms associated with cardiac distress. Cardiac beriberi could occur without neuritic or œdematous changes in the limbs. This was one of the most controversial subjects amongst our physicians, but in my humble opinion, a man either died of cardiac beriberi or completely recovered.

4. *Cerebral Beriberi.*—Frequently the first indication of cerebral beriberi was coma. Death was not uncommon.

A great deal has been written about the ætiology of beriberi. It appears to occur only in rice-eating people. I believe it is more than a vitamin B_1 deficiency, as pharmacological preparations of vitamin B_1 had little or no effect, while ordinary rice polishings—that is, the husk that has been removed in the polishing process—and ordinary commercial "Marmite" and "Vegemite" were the most beneficial. Thiamin given intravenously in large doses was more beneficial than tablets given orally. The administration of extra protein was also necessary. I considered that diet and not drugs is the remedy for beriberi. The majority of prisoners of war suffered from one or other form of beriberi, some slightly, others seriously. During the first year very few of us lost any weight—in fact, some increased in weight and girth. This was owing to the œdema; but at the end of 1942, when the men were put on to hard work, they rapidly lost weight.

Deficiency Scrotal Dermatitis.

Approximately four months after being taken into captivity we were confronted with a new condition known as deficiency scrotal dermatitis. Literally thousands of men were affected. Officers and other ranks suffered equally, but it was more prevalent in heavy duty men than in light duty men. The main features were bilateral œdema and erythema of the skin of the scrotum, which later swelled to an enormous size. In the early stages the condition was accompanied by an intolerable itch during the day, which was always worse at night. As a result of the œdema and the itching, the skin broke and a serosanguinous discharge was present. Large areas became red, raw and wet. The lesions were always symmetrical. As it progressed the condition spread into the groins, over the lower part of the abdomen, down the thighs and back to the natal cleft and anus and later involved the penis. Other accompanying changes were bilateral angular stomatitis, cheilitis and conjunctivitis, and an eczematous condition behind each ear. There were also various forms of glossitis and erythema of the palate and pharynx. The condition was so bad that hundreds of men became bedridden. A little later many of these sufferers complained of blurred vision, and examination revealed granular cornea, corneal ulcers, and later, involvement of the innervation of the retina, which was variously known as retrobulbar neuritis, amblyopia and neuroretinitis. Many of these men now have defects in their eyesight, even to blindness. I believe that those who came home with defective vision from this lesion will not improve, as nerve damage has occurred.

In the early stages the differential diagnosis lay between the following four conditions: (i) *tinea cruris*, known locally as dhobie's itch; (ii) chemical dermatitis; various applications were made to allay the itching; I have seen men actually paint the part with iodine, stating that they would prefer the pain to the itching; (iii) intertrigo; (iv) seborrhœic dermatitis.

The treatment of this syndrome caused a great deal of trouble. Available supplies of nicotinic acid, ascorbic acid and other pharmacological vitamin preparations and rice polishings had no effect. Ground-up peanuts promised well. There was no response to local treatment. However, we managed to obtain through the Japanese some ordinary commercial "Marmite" and "Vegemite". On the administration of an average dose of two drachms by weight, per may per day, the scrotal and skin conditions cleared up within fourteen days. Larger doses frequently set up diarrhœa. "Marmite" prevented, cured and controlled the skin and oral lesions, but it apparently had indifferent results upon the eye lesions.

This epidemic recurred on two further occasions: first, when we moved our camp in July, 1943, and again when we moved into the gaol in June, 1944. Each time our gardens were put out of bounds and we were deprived of greens. I believe we are justified in concluding that this syndrome known as scrotal dermatitis with its concurrent lesions was due to a deficiency of greens. Shortly after recognizing this in 1942, we commenced the manufacture of "grass extract", by extracting the juice of all available greens such as couch grass, hibiscus leaves and many other plants. This extract was given to "suspects" and patients regularly in daily doses of half a pint, and it relieved the early conditions and controlled the disease. But it is my firm conviction that "Marmite" and "Vegemite" stood alone in the treatment of these people.

There were no symptoms referable to the central nervous system, nor were there any gastro-intestinal symptoms, but owing to overcrowding and our inability to control hygiene, superadded infections were most common, the chief being scabies, tinea and skin diphtheria (this was prevalent during the whole period of our captivity).

Painful Feet.

In August, 1942, when we had the epidemic of scrotal dermatitis under control, a number of men began complaining of painful feet. This was another new disease for us. It was characterized by pain commencing in the ball of each foot at night time when the weight of the body was removed from the feet. Night workers complained of similar pains during the day. The pain varied from a dull ache, which gradually grew worse as the night wore on, to severe cramps and knotting of the muscles. Frequently these symptoms spread up to the calves of each leg. These men always obtained relief by getting up out of bed and walking about; men would sit for hours rubbing their feet.

Clinical examination revealed very little. The reflexes, both motor and sensory, remained intact, but in those subjects in whom the condition persisted for eight weeks or longer, the skin of the foot and the lower part of the leg became "thin" and had a glossy appearance, and occasionally there was evidence of atrophy of the interossei muscles of each foot. The condition was always bilateral. Many men became very depressed and exhausted from loss of sleep.

The condition was known by the men as "happy feet", the more scientific name being "pedialgia". A most interesting observation in these cases was that when any of these men developed pyrexia from malaria or any other disease the pain disappeared while the fever was on, only to recur when the temperature fell. This led to the application of infra-red heat in the treatment of these men, and to relief of the condition. (The filtration of the

heat was of interest.) The administration of "Marmite" and other available sources of vitamins had no apparent effect. The pain was almost invariably aggravated by any form of severe sickness, such as dysentery, malaria, scrotal dermatitis *et cetera*. Owing to local conditions, such as no pay and reduced rations for the sick by order of the Imperial Japanese Army, many of these men remained at work rather than report their illness. A cartel ship arrived at Singapore in 1942, and for about three months we lived on a good mixed diet of meat and vegetables; during this period the painful feet cleared up and many other deficiency diseases disappeared. I presume that the condition was due to protein deficiency.

The scrotal dermatitis syndrome and painful feet occurred mainly in men doing heavy duty. They were not common amongst the clerical staff and the indoor workers.

Œdema.

Mention has been made of the good diet in the latter end of 1942. My observation was that when the Japanese became amenable to reason there was always a catch somewhere. This time, after our men had been got into good condition by decent food, approximately 12,000 were removed in January, 1943, to work on the Burma-Thailand railway; reinforcements were sent in large numbers during the next few months.

During the three and a half years of our captivity most of us suffered from some form of œdema. During 1943 the health of the men in the Changi area, apart from malaria and dysentery, was fairly good. It was during this time that the Japanese gave us permission to keep poultry and establish a pig farm. We learned a lot about poultry, and we found that ducks are much more profitable than fowls. Poultry suffered from deficiency diseases just as we did. We could treat ducks, but we could not treat fowls.

For a period of approximately seventeen months the Japanese issued no meat and sent us in a variety of fish, "fresh" and sun-dried. The fish varied in length from one and a half inches to a twelve-foot shark.

During 1944 conditions deteriorated. They became worse after the fall of Saipan in 1944, and the poultry runs and the pig farm had to be destroyed and the rice was cut to twelve ounces per man per day. The pig farm was worth only an occasional two ounces of pork. More and more heavy labour was demanded by the Japanese. About this period the heavy duty ration was approximately 1,700 Calories per day, the light duty ration 1,200 Calories per day, and the no duty diet 700 Calories per day. By our own activities we were able to supplement the diet for the sick by as much as 500 Calories.

The clothing position became worse, and many men possessed only a ragged pair of shorts.

The constant heavy duty (up to fourteen hours a day), the low calorie diet and the extensive areas of skin exposed to the tropical sun, together with rapidly increasing malaria, recurrent dysentery, deficiency diseases and the ubiquitous vermin, caused the men gradually to lose weight—so much so that in January, 1945, the average weight of 220 men in my ward was 104 pounds, and mine was not the "skeleton" ward.

Tropical Ulcers.

Tropical ulcers made their appearance in our camp during the latter half of 1943. They usually occurred below the knees or on the forearm and hands, rarely in other areas. They started most frequently from a small punctured wound caused by bamboo or coconut palm fronds. In about twenty-four hours' time the part became painful, swollen and erythematous. Small hæmorrhagic vesicles appeared, and these broke about the third day, forming an ulcer which kept on extending in all directions. No tissue was immune. In many cases amputation of a limb was necessary, while other victims died. Many ulcers were aggravated by superadded infections, such as scabies, diphtheria *et cetera*, and by lack of treatment and forced labour. In some areas treatment was not available. I believe the main ætiological factors of tropical ulcers are: (i) malnutrition, (ii) debility owing to recurrent diseases, such as malaria *et cetera*, (iii) food and vitamin deficiency, undermining any remaining resistance to infection that the patient may have. My own observation was that if the ulcer could be cleaned, the part put at rest, and the patient fed on "Marmite" and sufficient protein, the ulcer would heal. Local treatment with sulphanilamide powder and sealing of the ulcer was most effective. In the absence of "Elastoplast", "Latex" was used. I do not think surgical treatment should be resorted to except as a last resort.

Pigmentation Dermatoses.

There were three outbreaks of pigmentation dermatoses, one in February, 1944. I believe this was a type of food poisoning from a particular type of bean of the calabar variety which the Japanese gave us to supplement our rations. About 25% of the men were allergic to this bean. The initial symptoms were the sensations of burning and pricking of the skin, particularly around the abdomen, the limbs and the face. These were followed in a few hours by œdema, which rapidly increased. The curious transverse furrows of the abdominal wall were characteristic in the initial stages, and if the face was involved the man would be unrecognizable in a few hours. Commonly the œdema of the skin of the abdomen felt to be about one inch in depth. This condition was followed in twenty-four hours by a mild papular rash, which was apt to involve the whole body. Later, in the next week, the skin began to peel, and in many cases the body was completely denuded of skin. In the more severe cases the whole of the thick skin of the palms of the hands and the soles of the feet came off almost in one piece and left a raw, bleeding surface. Other manifestations were swelling of the lips and tongue, splitting of the corners of the mouth, and multiple ulcers on the floor of the mouth, tongue, palate and pharynx. Multiple fissures occurred around the external anal margin, and many spread up inside the anus and involved one inch or so of the anal mucosa.

These patients presented an enormous problem in nursing and particularly in feeding. Blood counts in selected cases revealed a drop in the erythrocyte count to 3,000,000 per cubic millimetre, while the leucocyte count dropped to a few hundred per cubic millimetre. During the convalescent period and when the skin was regenerating, multiple boils appeared in various parts of the body, while in other cases superadded diphtheria infection in the ulcerated areas needed attention.

After a stormy convalescence many men were left with curious pigmented skin, most apparent around the margins of the hands and feet and also between the toes and fingers. The under-surface of the foot and the palms of the hands invariably remained whitish in colour, while the upper portions became a dirty brown. There was distinct margin between the discoloured areas. As the condition cleared, the impression was gained that ordinary dirt had been ingrained into the skin. Other more unfortunate patients had the pigmentation spread all over the body and the face; black, brown, red and white patches developed indiscriminately. In the dark races the skin became patchy white. A similar change was apparent on the eyebrows, the eyelashes, and the hair on the face. I think this pigmentation is permanent. The hair was bleached white in patches. During the course of the illness a rapidly running pulse was frequently observed and a rise in temperature up to 104° F. It was a long, dangerous illness with a stormy convalescence. When the bean was removed from our diet the condition cleared up and did not recur.

Another form of pigmentation occurred in the latter half of 1944. Men presented themselves with dark brown discoloration of the skin along the anterior and posterior axillary fold and in the cubital and popliteal fossæ. In this case the patients were never very ill, and they complained of a slight sensation of burning in the skin. Later the condition became painful. This discoloration came on within one to seven days. In the first couple of days the pigmentation could be made to fade on pressure, but from the third day onwards it would not fade and the parts

involved became extremely painful. If these patients were then taken into hospital, kept out of the sun, given mixed vitamins (the Japanese had given us a fairly large amount of mixed *A*, *B*, *C* and *D* vitamins) and grass extract to drink, the condition cleared in about a fortnight. These lesions were always symmetrical. There were rarely any symptoms referable to the digestive or nervous system. After their discharge from hospital these patients had to be kept indoors for a period of three or four weeks, or else the condition would rapidly recur.

It is possible that owing to the shortage of clothes and to the fact that the Japanese ration had been cut after the American capture of Saipan, this was another deficiency disease which was aggravated by exposure to the sun and possibly allied to pellagra, which occurred later in our area. It has been remarked on a previous occasion that no clothing was replaced by the Japanese, and in 1944 and 1945 a large number of the men possessed only a ragged pair of shorts or a G-string.

Pellagra.

In March, 1945, a condition resembling true pellagra first made its appearance. Many ætiological factors may have accounted for this disease. (i) Chronic debility was associated with malaria, many men having had the thirtieth to fortieth recurrence. (ii) Dysentery, amœbic and bacillary, was never completely wiped out, and frequently recurred in those who had had it. (iii) Beriberi was becoming worse. (iv) General malnutrition and emaciation were present. The daily diet at this time consisted of approximately 300 grammes of rice, eight grammes of "fresh" fish or dried fish (Japanese standard) and 300 grammes of vegetable. Occasionally maize in small quantities was mixed with our boiled rice. In April, 1945, of one hundred patients under my care, casually selected, there were 50 British, 35 Australians and 15 Dutch. The average weight was 104 pounds, the highest 152 pounds, and the lowest 87 pounds. The average amount of weight lost—that is, the difference between the weight prior to captivity and present weight—was 54 pounds, the highest was 86 pounds and the lowest was 21 pounds. (v) Clothing was almost completely lacking, and the men were constantly exposed to the tropical sun. It was observed that ninety of these people were doing heavy outdoor work for the Japanese. The other ten were hospital orderlies or indoor workers. (vi) Many of the men at this period were suffering from some form of dyspepsia and from one or more varieties of diarrhœa. Owing to the lack of food, many men on working parties used to "scrounge" some outside vegetable or other foodstuff. On many occasions it was a common thing for one half of the working party to come back suffering from acute diarrhœa and vomiting, apparently due to eating some poisonous food. (vii) Another factor in the emaciation of the men was the enormous number suffering from intestinal worms. This condition was so common that if any man complained of indigestion, abdominal pains or diarrhœa, we tested the fæces, and in the vast majority of cases one or other form of worms was found. (viii) Owing to the inroads of scabies and vermin, skin diseases and diphtheria *et cetera*, the constitution of the men was gradually undermined by sepsis.

This outbreak occurred at the end of February, 1945, and gradually began to reach its peak until relief came in August, 1945. It usually started with a brilliant red streak in the cubital and popliteal fossæ and sometimes on the dorsum of the foot and hand. In the first few days this erythema faded on pressure. Later fading did not take place. The skin became œdematous and painfully ulcerated. Here again the lesions were always symmetrical, and the parts first involved were those exposed to the sun. The condition progressed rapidly, so that at the end of four or five days, if the men were not treated and brought under cover, the whole of the body not covered by clothing was similarly involved. Œdema was most common on the feet and hands. In many instances large vesicles formed. These vesicles were filled with serous fluid. They often broke and formed ulcers of many shapes and sizes with sloughing edges. Under treatment the skin of the body and limbs gradually lost its red colour and became a dirty brown. The back of the neck in particular became darkened and the appearance resembled that seen on children who have a "high-water mark". These men, of course, were completely bedridden for a period of fourteen days or longer. In many anæsthesia developed, and in some paralysis of groups of muscles. These lesions also were always symmetrical. Angular stomatitis, chelitis, many and varied forms of glossitis and erythema of the palate were concurrent symptoms. As has already been mentioned, dyspepsia was such a common complaint that it cannot be stated definitely whether or not it was one of the symptoms of this condition.

The patients recovered quickly if they were taken into hospital, if their superficial lesions were dressed with bland dressings, and if they were given extra protein (by "extra" I mean probably one ounce of meat a day or one egg from our reserve supply) and "Marmite" or mixed vitamin therapy. Grass extract played its part. It was noted that if these men went back to their old conditions, they rapidly had a recurrence of their symptoms. There were no deaths, nor were any mental changes observed.

I think the surrender of Japan in August saved us from disaster of colossal magnitude. As the majority of men suffered from recurrent attacks of beriberi, malaria, dysentery *et cetera*, and as the Japanese "no duty" diet was 700 Calories per day, these men lost about one pound in weight per day in hospital. As soon as they obtained a diet of meat, butter, cheese, milk and ordinary European food they rapidly recovered and gained in weight. Of those who eventually left for home, the skin of only about five people remained pigmented. About ten had nerve lesions, the worst of which was spastic paraplegia. Except for these few cases, I do not think there will be any permanent effects. This condition I believe to have been a form of pellagra. Time does not permit a full description.

Sepsis.

It was noted after about six months of captivity that any abrasions or injuries of the skin rapidly became infected. For instance, an ordinary abrasion on the shin, which in civil life one would expect to clear up in a few days, frequently became incapacitating. Lymphangitis with swelling and pain in the lymph drainage area became manifest. I have seen patients with minor skin abrasions admitted to hospital within twenty-four hours with a pronounced degree of lymphangitis and adenitis.

Another condition which was frequently noted was a painful swelling, either superficial or deep, around some part of the leg. If it was superficial, obvious lymphangitis was noted, and if it was deep, lymphangitis was not apparent, but there was rapid enlargement of the inguinal glands, which became so painful that the patients became bedridden. In the majority of these cases there was no obvious external lesion or history of trauma. If the patients were not treated by rest and sulphanilamide, abscess formation followed rapidly.

The ease with which infection occurred and ran wild was in all probability due to debility and to lack of vitamin *A* complex. Thrombosis of the veins in various parts of the legs and thighs was most common.

Conclusion.

This talk has dealt only superficially with some of the medical problems which confronted us. Full detailed descriptions of the various diseases would occupy too much time tonight. I have only attempted to point out some of the things that will happen when diet and hygiene control break down. The disastrous eye conditions have already been mentioned in the *British Medical Journal* in December, 1945, and January, 1946. I have omitted reference to renal conditions. Prior to the war, I saw many natives with calculi in various parts of the urinary tract. Several times I saw calculi bigger than a cricket ball and occupying almost the whole of the bladder. Amongst the prisoners of war I do not remember having seen calculi, except in a few aged Dutchmen who were presented to us by the Japanese. There were, however, a

number of cases of transient infections of the urinary tract and a number of cases of non-specific urethritis. This in my opinion was usually due to degenerative changes in the mucosa following food and vitamin deficiency, with subsequent bacterial infection. By the uninitiated this might easily be mistaken for one of the types of venereal disease. The question of "wartime nephrosis" has been discussed by many other writers.

Reviews.

MEDICAL TREATMENT.

THAT well-known publication, a "Textbook of Medical Treatment", by Dunlop, Davidson and McNee, has reached its fourth edition.[1] The difficulty of compiling such a textbook "for students and practitioners" in these times of rapidly advancing therapeutic measures is recognized by the editors in their preface. It must be admitted that they have carefully kept the balance between discarding proven and well-tried methods and accepting recent innovations perhaps yet to be assessed by time. Thus the undoubted value of penicillin in many infections is recognized, but to avoid undue repetition a separate section is devoted to penicillin therapy to which frequent cross references are made throughout the book. Again, the therapy of thyreotoxicosis is dealt with very fully with particular reference to thiouracil, but a balanced view of the present controversy between some surgical and some medical schools of thought is presented to the reader. In passing, one mentions the subject of agranulocytosis referred to here, and, of course, in the section on blood diseases. The author of this section would appear to give full credit to "Sodium Pentnucleotide". Other authorities have not been so convinced of its value. No mention is made of the great reduction in mortality figures following the use of large doses of penicillin in agranulocytosis.

The section on tuberculosis, particularly pulmonary tuberculosis, is excellently presented. The indications for collapse therapy of various types are dealt with fully with the help of well-reproduced X-ray pictures. In fact, because early diagnosis and classification are essential to and inseparable from efficient treatment, this section as dealt with might well take its place in any medical publication, whether general textbook, diagnostic or therapeutic, so well is it set out.

Among the diseases of the respiratory system the difficult subject of the treatment of asthma is fully dealt with without the prejudice that is so often detected in writers on this subject. Several interesting case histories reveal the importance of the psychological factor, but perhaps most important there is a timely reminder that many factors, allergic, infective, nasal *et cetera*, often operate in the one person. The benefits of spa treatment are summed up in the delightful phrase as due to "the unintentional psychotherapy of the enthusiastic spa physician".

General practitioners especially will find the chapter on *status asthmaticus* particularly helpful.

In the chapter on diabetic coma we are particularly pleased to note the statement: "Too often quite insufficient doses of insulin are administered in this emergency from fear of inducing hypoglycæmia." This certainly would appear to have been the case with each generation of resident medical officers if we are to judge by the records of many teaching hospitals. Again: "It is insufficiently appreciated that five hundred units in the twenty-four hours, or even more, may have to be administered to patients in severe diabetic coma." After such excellent advice it is a pity to note the adherence to the intravenous administration of glucose, in view of evidence over the past two years, particularly from American workers, that such a procedure is not only unnecessary, but in fact may actually be harmful.

The section on psychotherapy in general practice adds greatly to the value of this book, and besides being highly informative is written in an attractive style. The chapters on the difficult subjects of alcoholism and drug addiction are helpful.

As in previous editions, there is a final chapter on technical procedures and oxygen therapy which must prove of great value to every busy practitioner and hospital medical officer. This edition concludes with a postscript on DDT and an appendix setting out a list of official preparations with their proprietary equivalents. A glance at the latter suggests that the "trade winds" from America do not blow as vigorously north of the Tweed as along the shores of Australia, and in this respect perhaps our Scottish colleagues are fortunate.

[1] "Textbook of Medical Treatment", by various authors, edited by D. M. Dunlop, B.A. (Oxon.), M.D., F.R.C.P. (Edinburgh), M.R.C.P. (London), L. S. P. Davidson, B.A. (Cambridge), M.D., F.R.C.P. (Edinburgh), F.R.C.P. (London), J. W. McNee, D.S.O., D.Sc., M.D. (Glasgow), F.R.C.P. (Edinburgh), F.R.C.P. (London), with a foreword by the late Professor A. J. Clark, B.A. (Cambridge), M.D., D.P.H., F.R.C.P. (London), F.R.S.; Fourth Edition; 1946. Edinburgh: E. and S. Livingstone, Limited. 9¼" x 6¼", pp. 942, with illustrations. Price: 30s. net.

TRAUMATIC CONDITIONS OF THE UPPER EXTREMITY.

"THE TRAUMATIC DEFORMITIES AND DISABILITIES OF THE UPPER EXTREMITY", by A. Steindler, is an important book, and comes from a surgeon whose name is well known in orthopædics.[1]

The plan of the work is excellent. In each section, a short systematic account is followed by illustrative cases, with photographs and skiagrams, and a statistical summary of the author's experience. References to the literature are given, and include the early classical contributions together with recent work. The book therefore will prove very helpful to surgeons faced with the late results of injury to the upper limb.

But the book is not beyond criticism. Many of the cases are unconvincing. Many of the photographs and skiagrams do not show what the text means them to show. Thus, Figure 181 shows the deformity "before", but not "after". Figure 182 shows it "after", but not "before". Figure 192 shows only forced extension "before", and only forced flexion "after". There does not appear to have been much improvement in this case, which is not surprising, because the skiagrams still show the bony fragment after the operation for its removal. Figure 285 gives no idea of the elbow and forearm movements. The operation was done for fracture of the type described by Monteggia in 1814, not in 1914. In Figure 294 the appearances of a dinner-fork deformity are most misleading, because the lighting comes from the finger-tip direction in the "before" photograph, and from the elbow direction in the "after" photograph.

B.J.W. is described as suffering from flexion contracture of the elbow, only three weeks after fracture of the lower end of the humerus. The treatment of three other cases by "capsulotomy" is hard to justify.

Figures 158, 406, 417 and 419 do not confirm the estimate of improvement given in the text. H.J. (Figure 412) does not appear to have benefited from her tendon graft, but this may be taken to illustrate the inherent difficulty of the procedure. Figure 194 shows a chip from the medial condyle of the humerus which is said to be within the elbow joint, and at the same time palpable under the skin. The skiagram shows the explanation of this contradiction. The chip is not within the joint, and the patient would have progressed just as well without the operation.

The anatomy is often obscure when the author deals with the nervous system and reflexes, as on page 362 and in Figure 322. On page 50 there is said to be no evidence of centripetal fibres in the sympathetic nervous system, in spite of the work of Gaskell and Langley over fifty years ago. Figure 199 is a most defective exposition of the anatomy of the antecubital region.

Notable omissions are the double-pedicle tube-graft in plastic work, and encouraged active movements in Sudeck's atrophy.

A final criticism may well be levelled at the use of English in this work. Words are used without regard to their precise meaning, as "pending" for during, and when the therapist in Sudeck's atrophy is advised to "establish" a trigger point, and then "follow it". Redundancies abound, as in "we advocate to remove the head by excision". Clumsy new words are coined, when old ones would have served. Thus we have "supinatory motion" for supination, "paroxysmatic" for paroxysmal, "necrotisation" for necrosis, "traumatism" for trauma, "mechanogenesis" for mechanism, "pathomechanics" for pathology and/or mechanism. In the half-page section on "Clinical Signs of the Sprains of the Wrist", there is only one brief sentence on clinical signs.

We feel a little mean in pointing out the smudges on a good picture, but it is our duty.

[1] "The Traumatic Deformities and Disabilities of the Upper Extremity", by Arthur Steindler, M.D., F.A.C.S., in collaboration with John Louis Marxer, M.D.; 1946. Springfield: Charles C. Thomas. 10" x 6¾", pp. 517, with many illustrations. Price: $10.00.

The Medical Journal of Australia

SATURDAY, AUGUST 3, 1946.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE NATIONAL HEALTH SERVICE BILL OF GREAT BRITAIN.

SINCE the beginning of April of this year several extensive references have been made in this journal to the National Health Service Bill of Great Britain and also to the attitude towards it that has been adopted by the medical profession in the Old Country through its organized body, the British Medical Association. The general attitude to the bill was expressed in unmistakable terms at the special Representative Meeting called to consider the matter on May 1 and 2, 1946. The meeting agreed on the need for improvement in and coordination of existing medical services, but disapproved of the methods proposed in the bill to achieve these ends. In the issue of May 25, 1946, we pointed out that while the representatives of the Association were discussing the bill at B.M.A. House, Tavistock Square, the Commons were discussing it at Westminster and gave it its second reading. Since that time the bill has been through the committee stage, a stage in which important changes are sometimes introduced into the provisions of proposed enactments. The results of the deliberations of the Commons in committee have been clearly set out in a leading article of *The Times*, a copy of which has been received by air mail. In order that readers may be familiar with the state of affairs in connexion with the bill on July 12, 1946, we propose to set out the chief facts described by *The Times* of that date.

The Times thinks that discussions in committee have been fruitful, and that though the bill has been changed in no fundamental respect, it has been improved in many minor ways. The Minister, Mr. Bevan, we are told, has been informative, persuasive and accommodating in his handling of the measure, and though he has yielded no ground on matters of principle, he has readily accepted suggestions for improvement in detail regardless of the quarter from which they have come.[1] The committee's discussions were "most perfunctory and confused" on the aspects of the bill affecting local government; matters such as the future lay-out of the maternity, child health, tuberculosis and mental health services were only lightly touched upon. It will be noticed that in the White Paper maternity and child welfare and mental health services are specially mentioned, but that no more than passing reference is made to tuberculosis. *The Times* goes on to point out that the Minister refused to be drawn into a general discussion about the future of local government. At the same time he rejected any suggestion that the new local health authorities should be permitted to delegate some of their duties to minor authorities, except in one instance for which provision is made in the bill. The way in which local authority medical services are to be managed under the new *régime* and the manner in which they will be related to the separately controlled hospital and general practitioner services are held to remain somewhat obscure; the same is thought to hold true of the method of control of health centres, which are to be maintained and staffed by local authorities for the use of medical practitioners not subject to their control. The Minister has given a clearer picture of the future management of the general practitioner and hospital services—neither service is to be centralized, as a first reading of the bill suggested. As an example of this decentralization we read that the filling of a vacancy in the public practitioner service will not be carried out by the central Medical Practices Committee, but by the local executive council after consultation with the recognized representative body of the local practitioners. The central committee will be charged with the duty of giving or withholding its approval of such appointments and will have the duty of watching over the distribution of doctors and of deciding which areas have too many and which too few. Readers will remember that the special Representative Meeting adopted a resolution in which the view was expressed that there should be no direction of a practitioner regarding the area in which he practises. Another feature of the bill to which the Representative Meeting objected strongly was the prohibition of the buying and selling of practices. The clauses relating to the "new offence" of selling the goodwill of a practice in the public service have been considerably "improved". Of what the improvement consists we do not know, but as the bill stands, it provides no channel of appeal for a practitioner against the decision of a special tribunal to exclude him from the public service either in one district or throughout the country. The committee has deleted from the bill the provision by which the doctor had the right to appeal to the Minister, "which was held to give insufficient protection", but refused to allow an appeal to the High Court. As *The Times* points out, there is a gap to be filled and Parliament will have to fill it. The Minister has made it clear in regard to health centres that he favours an experimental approach. Apparently there was a good deal of difference of opinion about this matter, but most people interested in the subject will agree with the view of the Minister; the same view, it will be remembered, has been expressed in medical discussions in this country. The Minister also, it appears, is "still unwavering in his opposition to a salaried service, but no less determined to introduce a basic salary into the remuneration of every doctor in the public scheme. . . ." This statement will be

[1] The White Paper, a "factual summary" of the provisions of the National Health Service Bill, was published *in extenso* in THE MEDICAL JOURNAL OF AUSTRALIA of April 13, 1946, at page 524.

accepted in the light of the Minister's remarks during the second reading of the bill that the medical profession was not "ripe" for a salaried service, and of his further statement that: "There is all the difference in the world between plucking fruit when it is ripe and plucking it while it is green." No sensible person will do other than conclude that the Minister will begin to waver in his opposition just as soon as he thinks that the fruit is ripe and no one will look on the basic salary as anything else in the mind of the Minister than part of the ripening process. The Minister did not comment on the Spens Report on the remuneration of medical practitioners. This is the report which was discussed at some length in these columns in the issue of June 8, 1946, and which has not yet been commented on officially by the Parent Body of the Association. *The Times* some weeks ago stated that the Spens Report would give Mr. Bevan something to think about. Many people in England and in the Dominions would like to know what he does think about it, and many too would be interested to know the British Medical Association's views about it.

The points that have been mentioned are those that will chiefly interest medical practitioners in this country. Mention has not been made of hospital plans. This is not our immediate concern. Suffice it to state that *The Times* comments on the enormous task that will face the twenty boards envisaged by Mr. Bevan as destined to serve the whole of England and Wales, and suggests that the size of the units may result in an administrative breakdown. Of this we shall no doubt hear more later.

The next news of happenings in Britain in regard to the National Health Service Bill will probably come with a report of the Representative Meeting of the British Medical Association that is scheduled to be held at the end of July, 1946.

Current Comment.

POISONING WITH ETHYLENE GLYCOL.

IT is remarkable what extraordinary substitutes for ethyl alcohol are drunk by members of that race of ours, cynically called by Mencken "homo boobiens". But while we must deplore the unnecessary tragedies thus caused, it is right to take all opportunities to learn what we can from them. Allied service records show numbers of cases of deaths from the imbibing of chemicals like methyl alcohol and ethylene glycol. It is likely that the ranks of Tuscany also have no reason for cheering on this account. Lieutenant-Colonel C. A. Pons and Major R. P. Custer, from the Army Institute of Pathology in Washington, have presented a valuable account of the laboratory data in a number of fatal cases of poisoning by ethylene glycol.[1] Ethylene glycol is used in anti-freeze mixtures in the cooling systems of internal combustion engines. It is colourless and odourless and is stated to have a pleasant bitter sweet taste and gives a warming sensation to the tongue and œsophagus when swallowed. The minimum lethal dose for human beings is estimated at about 100 millilitres.

Pons and Custer describe the case histories of some twenty patients who were all healthy soldiers. Since the circumstances in the field units where the pathological material was obtained did not always allow of detailed records, the histories are not always complete, but the general picture is clear. The amount consumed is uncertain, except in one case, in which it was about 200 millilitres. Most of the men looked shocked and usually had a subnormal temperature, and signs of involvement of the nervous system were observed in a number. These included ocular palsies and nystagmus, and, of greater significance, convulsions. In a few cases a bloody staining of the cerebro-spinal fluid was seen after lumbar puncture. All the patients died, most of them rapidly, the period elapsing between the drinking of the ethylene glycol and death ranging from twenty-two to forty-four hours. There was only one exception. One man survived five days in all, and his severe symptoms did not appear till two days had passed after the drinking of the poisonous fluid. It is stated that prior to this fatal indiscretion he had drunk several glasses of vodka, and it is open to speculation whether this had delayed the onset of other symptoms. In any case it is not suggested that vodka should be drunk as a precautionary measure if one feels impelled to drink ethylene glycol. This poison is remarkable in one respect, that it causes massive deposit of oxalate crystals in the renal tubules. It has been stated by other observers that death results from renal damage, and the recovery of two patients is recorded following decortication of one kidney. The evidence is not really convincing, as in the case of the value of decortication of the kidney in other conditions. Pons and Custer are much more impressed by the changes found in the nervous system which they regard as the true cause of death. Examination of autopsy material submitted to them showed swelling and engorgement of the kidneys, and in microscopic sections many crystals of calcium oxalate were found therein. The tubules in particular were seemingly packed with them, the sharp edges of the crystals appearing to pierce the walls in places. No degenerative changes were seen in the kidneys, except in the case of the man who survived some days. The whole central nervous systems were not available for examination, but hyperæmia was evident on naked-eye inspection in a number of cases. Examination of sections revealed definite and often striking degrees of damage, chiefly affecting the ganglion cells of the cortex of both cerebrum and cerebellum. The Purkinje cells of the cerebellum appeared to be specially vulnerable, as they were severely damaged. Examination of these sections by polarized light disclosed the presence of oxalate crystals in the brain and membranes as well as in the kidneys. These crystals were seen mostly within the small blood vessels and the perivascular spaces.

The authors have no doubt that the neural changes described are evidence of a true acute degenerative process. They suggest that the presence of oxalate crystals in the nervous system may be due to a slow building up of concentration of ethylene glycol in the central nervous system as happens with ethyl alcohol. There are strange features in the toxicology of this substance, and in view of its highly lethal nature its poisonous properties should be widely known.

[1] *The American Journal of the Medical Sciences*, May, 1946.

LIPOID PNEUMONIA.

ATTENTION was first drawn to the condition known as lipoid pneumonia in young children and infants, chiefly those in a debilitated state, and its recognition was due to pathologists. Some years ago it was pointed out in these columns that there was a certain risk of oily substances finding their way from the mouth or nose into the lungs of babies, especially if they were not vigorous. But since that time reports of cases in adults have accumulated, and it would appear that it is not only in little children that oil may, as their elders cheerfully say, "go down the wrong way".

W. A. Sodeman and B. M. Stuart have written an account of this condition in adults, and believe that it is of more than academic interest because it may simulate a number of other pulmonary conditions.[1] They distinguish between the types which show suggestive symptoms and those that are symptomless. Some of the former may be recognized only at autopsy, but others may be suspected from the radiological appearance of the lungs. The literature up

[1] *Annals of Internal Medicine*, February, 1946.

to date shows a preponderance of the asymptomatic group, though it appears that the references may not represent fairly the general experience of pathologists and clinicians. Sodeman and Stuart emphasize that the pathological nature of the lesion is a true inflammatory reaction directly attributable to the presence of oil droplets in the lungs; it is not sufficient to base a diagnosis on finding a few droplets and oil-laden macrophage cells due to agonal or terminal aspiration in acute pneumonic states of other ætiology. They state that lipoid pneumonia may simulate acute, recurrent or low-grade infections of the lungs, and even carcinoma. The X-ray film of the chest may show no definite changes in the lung fields, or some increase in the broncho-vascular markings of the lower lobes, but small areas may be seen suggestive of interstitial change or atelectasis. Actual nodulation or consolidation may be suggested in other cases in which the changes are more definite and extensive. Such pictures, ranging from the fine linear markings to consolidation, may persist for a long period and undergo little change; they are thus apt to give rise to confusion in diagnosis, since bronchiectasis, tuberculosis, malignant disease and other forms of inhalation pneumonia are simulated. These authors state that an acute bronchopneumonia which runs a protracted course is suspicious, as it may be of the lipoid type. They also quote a case in which the simulation of carcinomatous disease was so convincing that a pneumonectomy was performed, the lung being found after histological study to be the seat of chronic inflammatory changes due to aspiration of oil, without a trace of malignant degeneration. Irradiation has also been practised in such a case, owing to the same misconception.

With such warnings one naturally turns with interest to the subject of diagnosis. Little help is given by physical examination, as one might expect. The history is more helpful. There may be a coexisting disease, such as a nervous or nutritional condition affecting deglutition, and, more important still, there may be a history of either the long-continued instillation of oily drops into the nose, or of the constant taking of a mineral oil by mouth to correct a maladjustment of the colonic functions. In infants the issue is clearer, and the known administration of cod liver oil by mouth or paraffin drops into the nasal passages would suggest the possibility of pulmonary symptoms being due to the infiltration of oil in the lungs. But adults are prone to self-medication, whether on advice or not, and may easily omit to mention such a trifling matter, fraught with no apparent harm. Sodeman and Stuart mention the importance of searching for oil in the sputum, and advise allowing the sputum to stand, covering it with a cigarette paper to absorb the oil. As pathologists may nowadays find it very difficult to obtain the material for such a test they may prefer to make the microscope the arbiter. In the cases in which more serious diagnoses are suggested aspiration or biopsy may be justified. This account does not give us any sure guidance in diagnosis, but it brings out one moral clearly, that dogmatism based on partial evidence is dangerous. This dictum is not directed at the radiologist only, but at the ready reckoners of medicine of all kinds; we are all too prone to conclude that things are always what they seem.

NON-SPECIFIC URETHRITIS AND REITER'S DISEASE.

REITER'S DISEASE is the name given to a syndrome characterized by urethritis, conjunctivitis and arthritis. It was first described by Reiter in Germany in 1916 and has since been the subject of a voluminous literature. The triad of symptoms, of course, suggests a gonococcal cause, and as diarrhœa has been found to be a frequent initial symptom, the combination of diarrhœa, conjunctivitis and joint involvement also raises the question of dysentery. Reiter, however, produced evidence that the condition he described was quite distinct from these infections. Further experience has confirmed this by bacteriological and serological investigation. A well-documented case of this syndrome has been described by Bert L. Vallee, who also gives a review of the literature.[1] He points out that diarrhœa is a frequent early symptom, but is usually mild and lasts only some days. The urethritis and conjunctivitis are both characterized by a profuse yellow muco-purulent discharge, and appear from ten to thirty days after the subsidence of the enteric phase. The eye symptoms are usually confined to the conjunctiva, but keratitis, iritis and iridocyclitis may occur. The urethritis may not infrequently be followed by involvement of the bladder and prostate, and even of the kidney. It is of interest to recall that an abacterial pyuria is not uncommon as a separate entity, and a possible association with the so-called non-specific urethritis has been suggested. Returning to the description of Reiter's syndrome, the weight-bearing joints, sometimes including the spinal joints, become acutely inflamed and tender, and effusions containing lymphocytes but no demonstrable bacteria may occur. Other manifestations of the disease are a low-grade fever, vesicular and hyperkeratotic lesions of the skin and enlarged lymph nodes. The condition has been observed in many parts of the world, including Western Europe, America and the Pacific zone. There is no risk to life concerned, but residual changes may be observed in the eye and ankylosis of joints has occasionally occurred.

The ætiological problem set by this curious condition has not yet been solved. Vallee points out that it is certainly not gonococcal in origin, and that the most promising bacteriological work has been the finding of pleuropneumonia-like organisms in the urethral secretions. These pleomorphic organisms have also been found in various similar states in animals, and in the conjunctiva of normal mice. Similar organisms have been isolated by W. I. B. Beveridge from the urethra in cases of non-gonococcal or "non-specific" urethritis.[2] Such cases have been observed with greater frequency since the introduction of the sulphonamides in the treatment of gonorrhœa, especially as these drugs are ineffective in the non-gonococcal infections of the lower part of the urinary tract. W. I. B. Beveridge, W. D. Campbell and Patricia Lind repeated the findings of these pleuropneumonia-like organisms in the urethra of men suffering from non-gonococcal urethritis, and also demonstrated the occasional presence of the same bacteria in the genital tract of apparently normal women.[3] All this information put together seems to suggest a pattern, and it is an interesting hypothesis that we may have here the cause of "non-specific urethritis" and possibly, with metastatic symptoms added, that of Reiter's disease. This is, of course, pure hypothesis, but the evidence is somewhat in its favour. The fact that similar organisms are found in apparently healthy people is in no way antagonistic to such a suggestion. It has been said that Reiter's disease does not occur in women, but in view of the recent work this statement may perhaps be modified in the future. The method of transmission is a matter worth discussion. Reiter's disease might easily be transmitted by the same methods as the bowel-borne diseases, as well as possibly by the genito-urinary tract. What then about non-specific urethritis? Is it solely a venereal disease? Experience in both service and civil populations would lead us to believe that *per se* it usually is. But if abacterial pyuria is later found to be of similar bacteriological origin it would be difficult to deny the possibility of another route of infection than the genito-urinary tract. While we are speculating it is permissible also to ponder on the epidemic form of keratoconjunctivitis, which is believed to be due to a virus, and on the curious recent pandemic of mild diarrhœal disease in which no bacterial cause has been discovered so far. It is not suggested that these are of the same or even similar origin; but all these different infections are linked by certain related features which provoke us with our ignorance. It is most interesting how modern specific forms of therapy have stimulated inquiry into more exact ætiological classifications of disease, and have rearoused, or so we hope, our interest in the recognition of clinical pictures.

[1] *Archives of Internal Medicine*, March, 1946.
[2] THE MEDICAL JOURNAL OF AUSTRALIA, December 11, 1943.
[3] THE MEDICAL JOURNAL OF AUSTRALIA, February 9, 1946.

Abstracts from Medical Literature.

OPHTHALMOLOGY.

Persistent Primary Vitreous.

THE primary vitreous occupies Cloquet's canal. This canal runs through the centre of the posterior pole of the lens to the optic disk and originally contained certain structures which should atrophy but which may persist. A. B. Reese and F. Payne (*American Journal of Ophthalmology*, January, 1946) report thirty cases of persistence in premature and twenty in full-term infants. During the past forty-five years numerous reports have been made of cases in which the whole or a part of the *tunica vasculosa lentis* and the hyaloid sheath with its leash of vessels have persisted. In 30% of the present series the condition was noted before the sixth month; it was bilateral in 70%. Clinical appearance depended on the stage of cessation of normal involution and may be conveniently divided into four types: (*a*) central opacity of the cornea, usually associated with buphthalmos; (*b*) opaque tissue on the posterior surface of the lens; (*c*) the same, but more extensive with possible retinal detachment; (*d*) remains of the hyaloid vascular system. Clinically the first type may be confused with other forms of buphthalmos and with corneal changes due to intraocular inflammation; the second type may be confused with retinoblastoma; the third type may be confused with massive retinal fibrosis due to hæmorrhage at birth; and the fourth with newgrowth if hæmorrhage occurs. As early as the ten-millimetre stage of fœtal development the vitreous is being formed by ectodermal elements from the lens vesicle and invaginating optic vesicle and by mesodermal elements from the enclosed artery. Also at the forty-millimetre stage the vitreous vessels begin to atrophy, the parts nearest the retina becoming avascular first and forming the secondary vitreous. The atrophy should be complete by eight and a half months.

Diathermy in Glaucoma.

M. V. TRONCOSO (*American Journal of Ophthalmology*, March, 1946) has attempted to determine the physiological basis of diathermic methods in the treatment of glaucoma. For this he used a number of rabbits, and after operation kept a daily record of tonometric measurements. Vogt's method of multiple perforations in the region of the *orbicularis ciliaris* has no physiological basis, for this region has no part in the secretion of aqueous humour. It produced inflammation and atrophy of the muscle, but had little effect on the processes and none on the tension. Non-perforating diathermy also acted principally on the muscle, but it did have an indirect effect on the processes and lowered the tension temporarily. When multiple perforations were made over the processes they were destroyed sometimes to the accompaniment of exudate, hæmorrhages and lens changes. In some cases the eye atrophied. To determine the effect of the destruction of a small number of processes, two approaches were used, anterior through an iridectomy and posterior through the sclera. In all cases the tension, after remaining down for two to three weeks, returned to normal. None of the above procedures produced lasting hypotony. The author then opened the sclera, pushed a wire through the ciliary body until the tip appeared in the anterior chamber and turned on the current. In this way a combination of diathermy and fistula was obtained, and in this way only was the tension lowered for a long time.

Siderosis Bulbi.

J. BENDER SAMUEL (*The British Journal of Ophthalmology*, February, 1946) reports a case illustrating the value of interference in an advanced case of *siderosis bulbi*. The patient fired at a "dead" grenade twenty to thirty yards away. He felt the eye to be injured, but no X-ray examination was made. Vision remained the same for some time, then deteriorated to perception of light and the eye became painful. On examination a gross siderosis was present, and cornea, iris and lens were infiltrated with brown deposits, the lens being opaque and the tension raised. Miotics reduced the tension and X-ray examination revealed a foreign body measuring 2·4 by 1·0 millimetres. This was removed by the giant magnet through a scleral incision. A few weeks later the opaque lens was extracted extracapsularly and needled after the usual interval. The end result was vision correctable to $^{6}/_{12}$ and a slightly contracted field.

The Ishihara Test.

L. H. HARDY AND G. RAND (*Archives of Ophthalmology*, October, 1945) have investigated the reasons for failure to detect colour blindness by the Ishihara polychromatic plates. They assert that results obtained from these, with their low values and large patterns yielding extensive retinal images, must be applied with great caution to situations requiring the recognition of luminous objects which yield small but bright retinal images, for example, signals. Further, the amount and type of light used for illuminating the plates was found to influence results. For example, plate 4 was 100% efficient with some lights, only 43% with others. No analysis as to type or extent of defect in colour vision can be based on performance scores with these plates. Properly used they afford a good rough device for screening persons with defective red-green vision from the normal. The test should never be used alone, but always combined with some kind of lantern test, which more nearly approaches the conditions met with in industry and the services.

Dark Adaptation and Night Blindness.

F. W. PARKER, JUNIOR (*Archives of Ophthalmology*, May, 1946), presents the results of a study on dark adaptation in military personnel complaining of night blindness. The subjects were soldiers of the Army of the United States, serving in the Mediterranean theatre. Sixty-six subjects were examined with a Nagel adaptometer; careful ophthalmological examinations were made, and neuropsychiatric investigation was arranged when necessary. Thirty-nine of the subjects complained of moderate to extreme night blindness; fourteen were normal controls and thirteen were patients suffering from hepatitis. All the normal controls gave normal adaptation curves, and the final readings after forty-five minutes ranged from 50,000 to 150,000 Nagel units. Of none of them was the central visual acuity with correction less than $^{20}/_{20}$, and none had a refractive error of more than 1·00 diopter with homatropine cyclopiegia. Of the 39 patients who complained of night blindness, five who were moderately affected had normal adaptation, and three of these had more than −1·50 diopters of astigmatism. Six others gave normal readings, but complained of severe night blindness; detailed investigation revealed some incident that had caused the patient to believe that his night vision was inadequate; three of these subjects had moderate myopic astigmatism. The remaining 28 of the 39 patients gave low initial readings for dark adaptation. No cause for this could be found in five cases, and these subjects were considered to have true simple hemeralopia; four had an abnormal condition of the fundus; five were proved to be malingering; fourteen were psychoneurotic. Of the nineteen malingering or psychoneurotic patients, eleven had visual acuity of $^{20}/_{40}$ or less with correction, and twelve had refractive errors of more than 1·50 diopters. Of the thirteen patients admitted to hospital with the diagnosis of infectious hepatitis, one proved to be psychoneurotic. Six patients with chronic liver disease had subnormal dark adaptation; three of them were given vitamin *A* by mouth with pronounced improvement. Six patients with infectious hepatitis whose disease did not become chronic and whose period in hospital did not exceed fifteen weeks gave normal dark adaptation curves. In this group little or no improvement in dark adaptation was noted when massive doses of vitamin *A* were given. The author describes a test for malingering which utilizes the principle of opticokinetic nystagmus.

Nummular Keratitis and Ocular Brucellosis.

A. C. WOODS (*Archives of Ophthalmology*, May, 1946) states that in a previous paper with Guyton he held that there was reason to believe that in some instances nummular infiltrates in the cornea, the so-called nummular keratitis Dimmer, might be due to an infection with Brucella. He now presents further observations in support of this view. He reviews the literature and discusses the association of several authors' names with the term nummular keratitis, and states that attempts were made to identify the disease with a morphologically similar corneal disease of virus origin and finally with epidemic keratoconjunctivitis. The pertinent question whether all the cases described as nummular keratitis were manifestations of virus disease of the cornea cannot be answered with any assurance. The argument that some cases of nummular keratitis constitute a clinical entity, separate and distinct from epidemic keratoconjunctivitis, would rest on the demonstration of a specific, non-virus, ætiological agent. The author describes five cases in which nummular infiltrates of the cornea were

associated with serological and allergic evidence of an infection with Brucella. He also gives some details of an experimental investigation in which the corneæ of rabbits were inoculated with Brucella organisms and a similar clinical picture was reproduced. The use of the term Brucella nummular keratitis for this condition is suggested.

OTO-RHINO-LARYNGOLOGY.

Middle Ear Suppuration with Fistula.

M. TAMORI AND L. HIRSCH (*Annals of Otology, Rhinology and Laryngology*, June, 1945) state that in the past three years ten patients suffering from chronic ear suppuration with circumscribed labyrinthitis were operated on at the Illinois Eye and Ear Infirmary. All patients complained of dizziness and impaired hearing. Nine gave a positive response to the fistula test. A radical operation was performed on all ten patients; in two instances the endaural approach was employed. A visible fistula was found in only two instances. Two weeks' pre-operative hospitalization is recommended. All manipulations such as syringing and drops are strictly avoided. For three days before operation and for two days afterwards sulphathiazole is administered. All diseased tissue should be removed, but the area about the horizontal canal should be left undisturbed and the surgeon should avoid covering this region with any plastic flap or graft. The first post-operative dressing is done on the sixth day, and thence every other day the cavity is dusted with finely powdered sulphonamide drug and boracic acid.

Reflections on the Radical Mastoid Operation.

B. SEYMOUR JONES (*The Journal of Laryngology and Otology*, June, 1945) states that a survey of mastoid operations over many years and a close study of the eventual result has induced him, after practising skin grafting of the cavity for many years, to abandon that procedure entirely. The major reason given is that, unless regular supervision is undertaken, exfoliation of the epithelium occurs and later cholesteatoma formation and further bone erosion take place. While seeking to produce a dry ear, the author has also paid attention to the attainment of a pleasing cosmetic result. In preference to commonly used plastic procedures on the external auditory meatus, a single large flap is turned upwards, and this is claimed to avoid the formation of callus and fibrous tissue from the tegmen, so that narrowing and tunnel formation by approximation of the facial ridge and roof adjacent to the petrous base are avoided. The outer wall of the attic must be chiselled down until the roof of the cavity is quite smooth, and every effort should be made during this dissection to preserve the innermost portion of the meatal integument as part of the flap. To line the carefully smoothed mastoid excavation the author also uses a flap of periosteum dissected downwards off the mastoid process until it is attached only at its lower end. The facial ridge must not be covered at all, as this would lead to hyperplasia and narrowing of the meatal canal. In cases in which a lower flap is cut from the meatus instead of the upper flap, an incision is deliberately made in it to allow the facial ridge to project through. The cavity is packed tightly with "B.I.P.P." gauze, the outer end being brought out through a piece of rubber tubing which is pushed into the meatus. The posterior incision is closed. The packing is removed on the fourth day, and, after syringing with 1 in 80 carbolic acid solution, liquid "B.I.P.P." is poured in and a rubber tube is again placed in the meatus, but no further packing is inserted. The dressings are repeated every fifth day. Any exuberant granulations on the facial ridge are rubbed away. After the twelfth day acriflavine (1 in 1,000) gauze is tightly packed into the cavity and brings about further cleansing and represses granulations. For a persisting purulent secretion which may come from carious bone around the external semicircular canal, the application of 8% silver nitrate solution is effective. Two or three applications may be necessary at intervals of three weeks. On the second day after the application boric acid powder should be blown into the cavity. As soon as the mastoid cavity is dry, a trace of boric acid and resublimated iodine (0·75%) should be blown in once a fortnight, or where this is not tolerated "Zeroform" (tribromate of bismuth) may be substituted. In every case the patient should be seen by the surgeon at intervals of four months for removal of dead cells and other debris.

The King Operation for Bilateral Abductor Paralysis.

L. F. MORRISON (*Annals of Otology, Rhinology and Laryngology*, June, 1945) makes further observations on the King operation for bilateral abductor paralysis. The use of the omohyoid muscle for attachment to the arytenoid is a failure, since very little abduction is obtained through the action of this muscle. Severance of the interarytenoideus muscles is important, since, in spite of the adequate abduction obtained in the cord that is operated on, the cord not operated on may be pulled across towards the side which has been operated upon and so again narrow the airway. If the other side has subsequently to be operated upon, the resulting voice is likely to be of very poor quality. A further technical point of importance is the position of placement of the stay suture about the arytenoid. If this is set too far back from the vocal process, abduction of the arytenoid may be achieved, but a resultant internal rotation of the vocal process may neutralize much of the space gained in the airway. The site of fixation of the stay suture into the thyreoid ala is of less importance. The production of too wide a glottic space yields a poor voice. In other instances, even when the arytenoid is drawn right out against the thyreoid cartilage insufficient room is created, so that it may be necessary either to resect the arytenoid and abduct the remaining soft tissues, or to remove portion of the posterior edge of the thyreoid ala so that the abducted arytenoid may be sutured to the outer surface. A space of at least four or five millimetres is desirable. A nasal feeding tube is not necessary after operation. All the author's patients have been able to swallow without difficulty immediately after recovery from the anæsthetic. There is no fixed time of decanulation of the tracheotomy stoma. Attempts at decanulation should not be made prior to the fourth or fifth day, and then should be tested by partial and then complete plugging of the tube, which should not be finally removed until the patient has been found to be comfortable for forty-eight hours with the tube fully plugged. Some patients who have had a tracheotomy for a long time have difficulty in gaining confidence in breathing through the larynx again. In other cases actual œdema or failure to achieve the objects of the operation demands further delay. The author's conclusions are based upon a series of 23 patients, 22 females and one male. In twenty cases treatment was considered successful; failures occurred in three cases. The mortality was nil. For the most satisfactory operative course, convalescence and after-result, careful study of the patients' general state of health and the correction of hypothyreoidism and hypoparathyreoidism have been found to be highly important.

Recurrent Severe Hæmorrhage from the Nose and Pharynx.

FRANCIS B. SARGENT (*Annals of Otology, Rhinology and Laryngology*, September, 1945) is concerned with that type of massive recurrent hæmorrhage which is the result of erosion of a blood vessel beneath the surface and in which the exact source of the bleeding is not immediately evident. Invasion of surrounding connective tissue by infection and erosion of an arterial wall is the process leading to hæmorrhage. Four cases in which treatment was successful are described. In one instance there was bleeding from an ear and from around the tonsil of the same side. The internal carotid artery was tied and the patient recovered. In the second case, hæmorrhage associated with peritonsillar suppuration was controlled by ligature of the external carotid artery. In two cases hæmorrhage occurred from the nose and was diagnosed as coming from the sphenopalatine branch of the internal maxillary artery. In both these instances, after repeated packings had failed, the external carotid artery was tied with successful results. In a fifth case severe hæmorrhage occurred three days after the opening of a peritonsillar abscess and recurred three days later and a third time after the patient had been fifteen days in hospital. Ligation of the common carotid artery was asked for, but the surgeon tied only the external carotid artery, and left a loop around the internal carotid. After a further eight days another massive hæmorrhage occurred and was controlled by pulling on the loop around the internal carotid. That night the patient died of a further hæmorrhage, and at post-mortem examination it was found that the loop around the internal carotid artery had disappeared. In this case the continuance of hæmorrhage after ligation of the external carotid artery had been performed should have led to immediate ligation of the common carotid. The author concludes that it would appear that in the presence of evidence that the wall of a sizeable artery is eroded, immediate ligation is the only defensible treatment, regardless of the apparent success of less drastic measures.

Bibliography of Scientific and Industrial Reports.[1]

THE RESULTS OF WAR-TIME RESEARCH.

During the war a great deal of research was carried out under the auspices of the Allied Governments. It has been decided that a large proportion of the results of this research should now be released for general use.

The United States Department of Commerce, through its "Publication Board", is now issuing abstracts of these reports in the form of a "Bibliography of Scientific and Industrial Reports". The complete bibliography is being received in Australia, and extracts likely to be of interest to readers of this journal will be reproduced as far as practicable each week.

The original reports may be obtained in two ways: (a) Those marked with an asterisk may be obtained without cost on making application to Secondary Industries Division (Department of Post-War Reconstruction), Wentworth House, 203, Collins Street, Melbourne, C.1. (b) In other cases microfilm or photostat copies of the original report may be purchased from the United States through the Council for Scientific and Industrial Research Information Service. Readers desiring to avail themselves of this service should send the Australian equivalent of the net United States price to the Council for Scientific and Industrial Research Information Service, 425, St. Kilda Road, Melbourne, S.E.2. All other charges will be borne by the Council for Scientific and Industrial Research.

Further information on the subjects covered by the reports and kindred subjects may be obtained by approaching the Council for Scientific and Industrial Research Information Service, the Secondary Industries Division, Department of Post-War Reconstruction, or the Munitions Supply Laboratories (Technical Information Section), Maribyrnong, Victoria.

LAMANNA, CARL, *et alii*. The purification and crystallization of *Clostridium botulinum* type A toxin. Off. Pub. Bd., Report, PB 4184. 1945. 8 pp. Price: Microfilm, 50c.; Photostat, $1.00.

A highly toxic needle-shaped crystalline protein material has been isolated from a type A culture of *Clostridium botulinum*. Serological, pharmacological and electrophoretic data suggest that this material may be the pure toxin which causes a severe botulinus food poisoning. The method of separation, purification and crystallization was developed at Camp Detrick, Frederick, Md. This crystalline substance is probably the most toxic substance per unit of weight known.

BERGMANN, MAX, *et alii*. The penetration of vesicant vapours into human skin, to February 1, 1945. (OSRD Report 4855.) Off. Pub. Bd. Report, PB 5882. 1945. 41 pp. Price: Microfilm, 50c.; Photostat, $3.00.

Resolves the problem into the following four phases: 1. The development of precise analytical methods for measuring the amount of vesicant penetrated. 2. The construction of a suitable apparatus for applying vesicants, as vapours, to small areas of the skin. 3. A thorough test of the analytical methods and apparatus by means of appropriate model experiments. 4. The actual measurements of the rate of penetration of the vesicant vapours into human skin. Diagrams, tables and graphs.

BRUES, ALICE M. Body measurements of A.A.F. flyers (Graphic). (Army Air Forces. Engineering Divisions ENG-49-695-32-L.) Off. Pub. Bd. Report, PB 6963. 1943. 22 pp. Price: Microfilm, 50c.; Photostat, $2.00.

Graphic presentation of data on size of Army Air Forces flyers and flying clothing, based on data found in: Memorandum Report EXP-M-49-695-4-B, entitled "Facial Surveys of Aviation Cadets and other Flying Personnel"; Memorandum Report EXP-M-49-695-4-C, entitled "Anthropometric Data on Army Air Forces Flying Personnel"; and Memorandum Report ENG-49-695-32, entitled "Effect of Flying Clothing on Body Measurements of Army Air Forces Flyers". Tables included.

BRYSON, VERNON, DEMEREC, M., AND REISS, A. Second report on Contract No. W-29-057-CWS-35 between the War Department and the Long Island Biological Association, Inc. Off. Pub. Bd., Report, PB 6297. 1946. 11 pp. Price: Microfilm, 50c.; Photostat, $1.00.

Progress report on experimental infection of mice with pneumonia. Report discusses materials and methods used, establishment of penicillin controls, and quantitative study of the synergism of penicillin with a detergent (Aerosol). Synergism was most evident where Aerosol OT was used in a concentration of 1:50,000 combined with ·01 unit of penicillin per millilitre. Although either of these substances will permit the growth of bacteria when employed separately in the concentrations specified, no organisms are viable at the end of 24 hours when they are combined. Table.

CHAPANIS, ALPHONSE. The Luckiesh-Moss-Army Air Forces anoxia demonstration charts, types AAF-1 and AAF2. (Army Air Forces. ENG-49-695-12N.) Off. Pub. Bd., Report, PB 6949. 1943. 12 pp. Price: Microfilm, 50c.; Photostat, $1.00.

Two new visual charts, known as the Luckiesh-Moss-Army Air Forces anoxia demonstration charts, types AAF-1 and AAF-2, have been developed for demonstrating the effects of anoxia on contrast sensitivity under dim illumination. A descripton of these charts and a digest of earlier work on the subject appear in Appendix 1. Methods for illuminating the chamber to the proper level are illustrated in Appendix 2. Instructions for administering the tests are given in Appendix 3. Illustrations of type 1 chart, diagram of lighting plans and references included.

CHAPANIS, ALPHONSE. Night vision tests and the night vision testing programme in the Army Air Forces. (Army Air Forces. Engineering Division. ENG-49-695-37c.) Off. Pub. Bd., Report, PB5140. 1944. 25 pp. Price: Microfilm, 50c.; Photostat, $2.00.

The purpose of this report is: 1. To point out some errors in the statistical analysis of night vision test data used in the A.A.F. School of Aviation Medicine Research Report No. 1, Project No. 213. 2. To summarize night vision test data collected recently by the Aero Medical Laboratory and to show that these data and the School of Aviation Medicine data are actually in essential agreement. Tables.

CHAPANIS, ALPHONSE. Results with the Luckiesh-Moss-Army Air Forces anoxia demonstration chart, type AAF-1, at simulated altitudes of 15,000 and 16,000 feet. (Army Air Forces. Engineering Division, ENG-49-695-12-0.) Off. Pub. Bd., Report, PB 5133. 1943. 9 pp. Price: Microfilm, 50c.; Photostat, $1.00.

This report includes a summary of the results obtained with the chart, described in Report ENG-49-695-12N, as used by one hundred men at each of the two simulated altitudes, 15,000 and 16,000 feet. Tables.

COON, JULIUS M., *et alii*. Phosgene: Special studies to January 2, 1945. (OSRD Report 4637.) Off. Pub. Bd., Report, PB 5907. 1945. 39 pp. Price: Microfilm, 50c.; Photostat, $3.00.

The report presents results and conclusions from experiments designed to clarify the relation between L(Ct) and exposure time of animals to CG for a short time and for various concentrations.

DILL, DAVID B. Aeroembolism. (Army Air Forces Engineering Division. ENG-M-49-696-1C.) Off. Pub. Bd., Report, PB 5141. 1943. 24 pp. Price: Microfilm, 50c.; Photostat, $2.00.

The purposes of this study were: 1. To analyse changes in physiological states that occur in aeroembolism. 2. To outline plans for attacking the problems of aeroembolism. Dr. Melvin H. Knisely, of the Department of Anatomy, University of Chicago, who conducted the study, found that subjects experiencing aeroembolism have prolonged spasms of the arterioles of the connective tissues and striated muscles which completely shut off their blood supply for long periods of time. No intravascular agglutination of blood cells was revealed by this study, and no gas bubbles in or adjacent to blood vessels were seen.

DILL, DAVID B. Aeroembolism. (Army Air Forces. Experimental Engineering Section. EXP-M-54-653-44A.) Off. Pub. Bd., Report, PB 5114. 1941. 3 pp. Price: Microfilm, 50c.; Photostat, $1.00.

Aeroembolism is known as bends, decompression illness, aeroemphysema, caisson disease or aeropathy. It is attributed to the formation of gas bubbles in the tissues of the body during exposure to low pressure and to the expansion of these bubbles with time or with further decreases in pressure. Protection against aeroembolism may be gained by reducing the nitrogen in the body; its complete elimination is not required.

DILL, DAVID B. Desert water requirements. (Army Air Forces. Experimental Engineering Centre. EXP-M-49-696-2.) Off. Pub. Bd., Report, PB 5143. 1942. 9 pp. Price: Microfilm, 50c.; Photostat, $1.00.

Report of observations on the supply of water to personnel of the Army Air Forces in desert operations. It was concluded that flight personnel in desert operations require less water than ground crews.

DILL, DAVID B. Effects on pilots of high centrifugal forces. (Army Air Forces. Experimental Engineering Section. EXP-M-54-853-41.) Off. Pub. Bd., Report, PB 5111. 1941. 6 pp. Price: Microfilm, 50c.; Photostat, $1.00.

This report contains instructions for standing crew members and seated personnel in how best to resist the effects of high centrifugal forces. Special measures for increasing resistance to centrifugal forces and general precautionary measures are included, as well as an illustration of the best position to withstand high acceleration.

[1] Supplied by the Information Service of the Council for Scientific and Industrial Research.

ELBERG, S., EDWARD, E. P., AND SWANSON, R. W. A selective medium for the isolation of *Brucella suis*. Off. Pub. Bd., Report, PB5520. 1945. 6 pp. Price: Microfilm, 50c.; Photostat, $1.00.

A bacteriological medium which may prove to be of value in isolating the causative organisms of the swine or porcine type of brucellosis or undulant fever from fæcal samples or other heavily contaminated specimens, is described as the result of preliminary studies carried out at Camp Detrick on this disease. This report covers studies which are rather narrow in scope. However, it is felt that this medium may prove of value as a tool for other research workers. The medium contains, in addition to commonly used constituents, small amounts of tyrothricin and sodium azide.

GEILING, E. M. K., *et alii*. Toxicity and incidence of lipid pneumonia upon inhalation of automobile oil cloud (S. A.E. No. 10), to August 12, 1942. (University of Chicago Toxicity Laboratory Report 9.) (OSRD Report 809.) Off. Pub. Bd., Report, PB 6578. 1942. 9 pp. Price: Microfilm, 50c.; Photostat, $1.00.

Report of experiments to determine the toxic effects and incidence of lipid pneumonia in mice exposed to an automobile cloud in order to investigate the potential hazard of lipid pneumonia for personnel compelled to work in mineral oil fogs dispersed as clouds to screen military objectives from enemy aircraft. After 100 consecutive days of exposure to an oil cloud containing 132γ of oil/l. there is no significant reaction to oil in the lungs. The aspirated oil is not toxic, since there was no increase in spontaneous deaths. Chemical studies revealed that after the 100-day period of exposure the lungs of twenty mice contained 33 milligrammes of oil, an average of 1·65 milligrammes per mouse. This amount of oil represented only 0·4% of the total weight of the lungs of each mouse. The quantitative data from the chemical study, together with the observations of the lack of any significant reaction of lungs to the oil *in situ*, seem to indicate that exposure to the oil cloud had no significant detrimental effect. Report contains a table showing results of the analysis, and a bibliography.

HALL, FRANK G. Aeroembolism. (Army Air Forces. Experimental Engineering Section EXP-M-49-696-1A.) Off. Pub. Bd., Report, PB 5142. 1942. 5 pp. Price: Microfilm, 50c.; Photostat, $1.00.

Details of a case of collapse resulting from severe aeroembolism are reported and suggestions applicable to such situations in the future are given. Also included is the log of a simulated flight to 35,000 feet.

HALL, FRANK G., AND WILSON, JOHN W. Alveolar air compositions. (Army Air Forces. Engineering Division. ENG-49-696-42-F.) Off. Pub. Bd., Report, PB 5161. 1944. 6 pp. Price: Microfilm, 50c.; Photostat, $1.00.

Report of experiments carried out at Peterson Field, Colorado, to determine the effect of acclimatization of individuals to an altitude of 6,200 feet upon the composition of alveolar air. It was concluded that acclimatization of individuals who have been living at or near sea level to approximately 6,200 feet, produces a significant change in alveolar air composition. Also, such acclimatization increases the individuals' altitude-tolerance ceiling. Incidental to the alveolar air studies, observations were made on the symptoms of the subjects during and after the altitude flights. Tables are given showing: 1. The age and period of acclimatization of the subjects; the maximum altitude attained by each and the subjective symptoms of the individuals after the period of anoxia. 2. The subjective and objective symptoms experienced by subjects while breathing atmospheric air at simulated altitudes between 12,000 feet and the highest altitude which the individual could tolerate.

HALL, J. F., JUNIOR. Effect of sulphonamides on the blood pigments, oxygen and carbon dioxide capacity of dogs and humans. (Army Air Forces. Experimental Engineering Section EXP-M-49-695-5A.) Off. Pub. Bd., Report, PB 6942. 1942. 19 pp. Price: Microfilm, 50c.; Photostat, $2.00.

Report of a group of forty-six experiments upon three human subjects and eight dogs. Procedures, results and conclusions are given. In view of the negligible effects of sulphadiazine on the oxygen and carbon dioxide capacity of the blood and the relatively high blood concentrations attained with single oral doses, it is recommended that this sulphonamide be administered by medical officers to wounded or injured personnel who may be subjected to aircraft flight. Tables and a graph are included.

HALL, J. F., JUNIOR. Effects of certain drugs and blood loss upon altitude tolerance. (Army Air Forces. Experimental Engineering Section EXP-M-49-695-5B.) Off. Pub. Bd., Report, PB 6943. 1942. 11 pp. Price: Microfilm, 50c.; Photostat, $1.00.

Tests on dogs were conducted to investigate the effects of blood loss and drugs (morphine and sulphanilamide) upon the ability of animals to withstand exposure to altitude, as indicated by change in pulse rate, change in respiration rate, and reduction in oxygen-carrying capacity of the blood. Experiments are tabulated and details of procedure are given. Appendices contain graphic presentation of results. Conclusions reached in this experimental study are not necessarily valid when applied to human subjects.

HEIM, J. W. Occurrence of aeroembolism. (Army Air Forces. Experimental Engineering Section. EXP-M-54-653-44.) Off. Pub. Bd., Report, PB 5113. 1941. 5 pp. Price: Microfilm, 50c.; Photostat, $1.00.

Ninety-one men were exposed to altitudes of 30,000, 35,000 and 40,000 feet while breathing pure oxygen in the high altitude chamber for various periods of time and at various rates of climb. Symptom data were recorded. It was noted that the incidence of symptoms was reduced in subjects who breathed oxygen while exercising during ascent, and throughout the study at 40,000 feet it was effective in preventing the appearance of symptoms.

HEIM, J. W. The present status of the carbon monoxide problem in aircraft. (Army Air Forces. Experimental Engineering Section. EXP-M-54-653-116.) Off. Pub. Bd., Report, PB 6925. 1942. 37 pp. Price: Microfilm, 50c.; Photostat, $3.00.

A review of incidence of carbon monoxide, permissible concentrations, measuring instruments and warning devices, test procedures, a remedial action and detection in man after exposure. It is recommended that the development of a suitable type indicator be vigorously pursued as well as a field method of blood analysis for carbon monoxide. Results of tests are given in tables.

HENRIQUES, F. C., JUNIOR, *et alii*. An experimental investigation of the physiological mechanisms concerned in the production of casualties by flame-thrower attack. (OSRD Report 4029.) Off. Pub. Bd., Report, PB 4208. 1944. 44 pp. Price: Microfilm, 50c.; Photostat, $3.00.

This is a progress report to August, 1944, under contract 9-93, NDCrc-169 with Harvard University. The physical data are contributed by F. C. Henriques, junior, and the physiological and pathological data by A. R. Moritz, F. R. Dutra and J. R. Weisiger. This report covers initial observations on the cause of death in animals exposed for short periods of time to high circumambient temperatures. Such observations, uncomplicated by the presence of asphyxial gases, hot carbon particles *et cetera*, are necessary in order to assess the lethal action of flame from various fuels burning in partially enclosed spaces. The authors have carried out their studies in a quantitative manner, and evaluated their results in terms of the caloric bombardment (cal./gm. biot.) received by the animal. Report contains graphs, diagrams and tables.

HENRIQUES, F. C., *et alii*. The mechanism of cutaneous injury by mustard gas. An experimental study using mustard prepared with radioactive sulphur, to November 10, 1943. (OSRD Report 3620.) Off. Pub. Bd., Report, PB 5930. 1944. 121 pp. Price: Microfilm, $1.50; Photostat, $9.00.

This report presents in full the results of over three years of study. Extensive evidence points to the conclusion that it is the rapidly completed chemical combination of mustard with one or more cellular proteins (bound mustard) that is responsible for cellular injury. Among contributions of this work are pathological studies of a complete, progressive series of mustard burns in man, obtained by biopsy, and a comparison with a similar series from the pig and rabbit, data on the absolute rates of penetration in man, the pig and rabbit, and data on the distribution of fixed mustard in the epidermis and corium and in the nuclear tissue of the epidermis. Although the main conclusions of the study are discouraging, so far as the hopes of obtaining a specific antidote for mustard is concerned, it is of great value in the vesicant problem, in that it establishes a precise frame of knowledge relative to the limits and requirements both for decontamination and specific therapy. Diagrams, photographs, tables and a bibliography are included. This is a progress report under contract with Harvard University. See OSRD Report 3620-A (PB 5917) for final report on these studies.

KIBLER, A. L. The after effects of chloracetophenone. (Edgewood Arsenal Technical Report 133.) Off. Pub. Bd., Report, PB 6278. 1933. 19 pp. Price: Microfilm, 50c.; Photostat, $2.00.

Pertinent data regarding the physiological effects of CN have been collected and recorded in this report. Opinions regarding the after effects of CN have been quoted from authorities on the physiological effects of toxic gases, and statements have been obtained from personnel at Edgewood Arsenal known to have been exposed to high concentrations of CN for long periods of time. The primary effect of CN on the eyes, causing pain and intense lacrymation, subsides very soon after removal from the vapours. In some cases exposure to CN vapours for a considerable time causes a skin rash, but this rash disappears after a few days and leaves no after effects. Photographs.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on June 20, 1946, at the Royal North Shore Hospital of Sydney. The meeting took the form of a number of clinical demonstrations by members of the honorary medical staff of the hospital. Part of this report appeared in the issue of July 27, 1946.

Patent Ductus Arteriosus.

DR. B. T. SHALLARD showed four patients suffering from patent *ductus arteriosus*. The first was a female patient, aged fifteen years. She had suffered from dyspnœa on exertion since infancy, from precordial pain induced by excitement for one month and from palpitation. She was not cyanosed. The systolic blood pressure was 130 millimetres of mercury and the diastolic pressure 60 millimetres in the right arm; the figures obtained in the left leg were respectively 165 and 95 millimetres. A thrill was palpable continuously throughout systole and diastole in the upper part of the precordium, and thoughout systole only in the mitral area. The apex beat was heard in the fifth left intercostal space, four and a half inches from the mid-line. A harsh murmur at the base was continuous throughout systole and diastole. An electrocardiogram revealed right axis deviation. Ligation of the patent ductus was undertaken on March 7, 1946. The murmur was obliterated at operation, but returned soon afterwards; the thrill returned one week later. The temperature was "hectic" for nearly two months, and at the time of the meeting it was still irregular. The blood pressure in both right arm and left leg was 115 millimetres of mercury, systolic, and 65 millimetres, diastolic. An electrocardiogram revealed left axis deviation. Myocardial degeneration was present.

The next patient shown by Dr. Shallard was a female, aged nine years, suffering from dyspnœa on exertion, easy fatigability and cyanosis after exertion. She was stunted in growth. The systolic blood pressure was 105 millimetres of mercury and the diastolic pressure 65. The apex beat was heard in the fifth left intercostal space, three and a half inches from the mid-line. A systolic thrill was present over the whole of the precordium. A harsh murmur was continuous throughout systole and diastole, loudest at the pulmonary area. X-ray examination revealed signs of congestion of the cardiac type; the heart was not enlarged.

Dr. Shallard then showed a male patient, aged nine years, who was easily fatigued and frequently coughed. He was not cyanosed. The systolic blood pressure was 135 millimetres of mercury and the diastolic pressure 60 millimetres, in the right arm; in the right leg the figures obtained were 155 and 55 respectively. A pistol-shot sound was heard on auscultation over the femoral and radial arteries. A precordial thrill was palpable. The apex beat was in the sixth left intercostal space in the anterior axillary line. A murmur was continuous throughout systole and diastole and was heard at the pulmonary area. The patient's temperature had been elevated intermittently since his admission to hospital. An electrocardiogram revealed right axis deviation. X-ray examination showed that the heart was much enlarged.

The next patient shown by Dr. Shallard was a male, aged three years, who suffered from dyspnœa on exertion. The systolic blood pressure was 90 millimetres of mercury and the diastolic pressure nil. A thrill was present over the precordium, and a rough, blowing systolic and diastolic murmur was heard. On X-ray examination the cardiac shadow was slightly larger than usual, and globular. The appearances suggested a congenital heart lesion.

Patent Interauricular Septum.

Dr. Shallard then showed two patients suffering from patent interauricular septum. The first was a female, aged six years, who suffered from dyspnœa, easy fatigability and unpleasant turns with a smothering sensation. She was stunted in growth, but no clubbing of the fingers or toes and no cyanosis was present. The systolic blood pressure was 105 millimetres of mercury and the diastolic pressure 65. A precordial bulge was observed. A thrill and systolic murmur were noted in the second left intercostal space, a little distance out from the sternal border. The second pulmonary sound was audible but weak. X-ray examination revealed a dilated pulmonary artery. An electrocardiogram revealed right axis deviation; the *ST* wave in Lead III was depressed, and the *T* wave was diphasic.

The next patient was a male, aged fifty-nine years, who had suffered from dyspnœa without cyanosis for eighteen months. No abnormal physical signs (murmurs *et cetera*) were present. The systolic blood pressure was 130 millimetres of mercury and the diastolic pressure was 90. X-ray examination revealed dilatation of the pulmonary artery. An electrocardiogram revealed right ventricular strain.

Patent Interventricular Septum.

Dr. Shallard then showed a female patient, aged eight years, who tired easily. She was not cyanosed. A systolic murmur was present, best heard medially. The apex beat was in the third left intercostal space. The systolic blood pressure was 120 millimetres of mercury and the diastolic pressure 80. X-ray examination revealed that the heart was globular in shape; right and left ventricular hypertrophy was present. There was no conus projection.

Another patient with a patent interventricular septum was a female, aged eighteen years. She had suffered from dyspnœa on exertion for two years. She was not cyanosed. The systolic blood pressure was 140 millimetres of mercury and the diastolic pressure was 90. A systolic thrill was palpable in the second left intercostal space. A harsh, blowing systolic murmur was heard at the pulmonary area, with a loud second sound. X-ray examination revealed some left ventricular hypertrophy. An electrocardiogram disclosed no abnormality.

Eisenmenger's Syndrome.

Dr. Shallard then showed a female patient, aged twenty years, who had suffered from dyspnœa for three years and from œdema of the ankles for four months. Pain in the chest and distress were experienced on exertion. She was slightly cyanosed, and slight clubbing of digits was present. The systolic blood pressure was 125 millimetres of mercury and the diastolic pressure 100. The heart was enlarged to the left. Gallop rhythm was present, especially at the base. No thrills or murmurs were detected. The liver was enlarged. Fluoroscopic examination revealed hypertrophy of the right side of the heart; the pulmonary artery was prominent. No hilar dance was observed. An electrocardiogram revealed right axis deviation; the *P* waves were large, the *P-R* interval was excessive, and the *ST* waves were deformed in Leads I and III.

Lutenbacher's Syndrome.

Dr. Shallard finally showed a female patient, aged five years, who suffered from dyspnœa and from attacks of loss of consciousness. She was cyanosed. The liver was enlarged to below the umbilicus, and there was an area of shifting dulness in the abdomen. Pitting œdema of the ankles was present. The heart was greatly enlarged; a thrill was present at the apex, and a loud systolic murmur was heard at all areas. X-ray examination revealed that the heart was much enlarged to both sides and irregular in outline: congestive changes were present in the left lung. An electrocardiogram revealed extrasystoles, a pronounced myocardial defect, and abnormal auricular and ventricular complexes.

Carcinoma of the Naso-Pharynx with Abducent Paralysis.

DR. DOUGLAN ANDERSON showed a male patient, aged fifty years, who had become ill one year earlier with right-sided earache and severe headache. He had noticed a thumping sound in the right ear for several months before the onset of his illness. About six months after he became ill, there developed on the right side of his body abducent palsy with pseudoptosis, analgesia in the area supplied by the trigeminal nerve, middle-ear deafness, and paralysis of the palate, pharynx, larynx and tongue. No facial palsy was present. The paralysed side of the tongue wasted. Three months later he had an hæmoptysis and was aware of a foul odour in his air passages; he had by then lost over two stone in weight. A friable neoplasm was discovered in the naso-pharynx, near the aperture of the right auditory tube. One month prior to the meeting it had been treated by deep X rays. The mass had disappeared, as had the patient's symptoms, except that the wasted side of the tongue had not yet recovered. Dr. Anderson remarked that it seemed curious that the abducent nerve, the course of which was wholly intracranial, should be affected by a neoplasm which appeared to be wholly extracranial; but he pointed out, by reference to a skull, by how little the nerve was removed from the pharynx as it passed by the *foramen lacerum*.

(*To be continued.*)

NOTICE.

THE General Secretary of the Federal Council of the British Medical Association in Australia has announced that the following medical practitioners have been released from full-time duty with His Majesty's Forces and have resumed or will resume civil practice as from the dates mentioned:

Dr. Craig A. Horn, 137, Herries Street, Toowoomba, Queensland (July 15, 1946).
Dr. R. Hertzberg, 9, Butler's Road, Hurstville, New South Wales (August 5, 1946).
Dr. R. M. Hollingworth, Auburn Street, Moree, New South Wales (June 14, 1946).
Dr. E. W. Lee, "Locarno", 141, Macquarie Street, Sydney (July 8, 1946).
Dr. R. F. K. West, 460, Pacific Highway, Lindfield, New South Wales (June 18, 1946).

Medical Societies.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING of the Medical Sciences Club of South Australia was held at Adelaide on May 3, 1946.

Motor Mechanisms in the Central Nervous System.

PROFESSOR A. A. ABBIE read a paper on the motor mechanisms in the central nervous system. He said that the motor executives of the central nervous system were the voluntary muscles, which could do no more than contract or relax; and the whole object of nervous control was to secure contraction (by excitation) or relaxation (by inhibition) of the appropriate muscles in the proper sequence, to the desired extent, at the correct speed and with the necessary force.

There were three levels of control—spinal, mid-brain and forebrain. At the spinal level coordination of contraction and relaxation was effected through the reciprocal innervation demonstrated by Sherrington. In all cranial motor mechanisms, however, the vestibulo-cerebellar apparatus came into play through the vestibulo-spinal tract to maintain the basic biological posture such as was exhibited in decerebrate rigidity when higher controls were removed.

In the mid-brain the excitatory mechanism resided in the tectum and its effects were mediated through the tecto-spinal tract, as was disclosed by stimulating the optic lobes in reptiles and birds. The inhibitory mechanism was in the cerebello-dentate-rubro-spinal circuit.

Probably all mammals had a measure of forebrain control of movement, although it might be difficult to demonstrate in some of the more primitive. Control was first restricted to head and forelimbs, but was later extended to the hind-limbs—as shown in the marsupials and monotremes and more primitive *Eutheria*. The transverse, inverted, representation of the body in the cerebral cortex was probably to be explained as the rotation of an originally antero-posterior representation, the rotation being due to expansion of more frontal areas.

In the human, the excitable precentral cortex included a number of the Brodmann formations—8, 6 *plus* 44, 4a and 4γ. Area 8 had an internal granular lamina; the remainder were all agranular, but only 4γ had the giant cells of Betz in lamina V. In area 8 only the posterior end of the middle frontal gyrus was excitable, producing conjugate deviation of the head and eyes towards the opposite side. Areas 6 and 44 seemed to be concerned with coordinated whole movements; area 44 acting thus for the muscles of the mouth and larynx and, in the dominant hemisphere, being the "motor speech area" of Broca. Passing posteriorly through areas 4a and 4γ, stimulation produced responses more and more restricted until individual muscles alone contracted; the threshold, too, was lower there. Inhibition from the cerebral cortex was effected by several areas—8, 4s, 2s and 19. Of these, 4s seemed to be the most important for the present purpose.

The excitatory and inhibitory impulses all seemed to pass through the corresponding pyramidal tract. The Betz cells only contributed about 3% of the pyramidal fibres and the whole of the precentral gyrus not more than 40%. The origin of many of the others was uncertain. About 10% of the pyramidal fibres remained uncrossed. The point of termination of pyramidal fibres was in dispute. Either they ended directly on the anterior horn cells or they ended in relation to internuncial neurones in the posterior horn. Both views had equal support from different observers; probably both were partly correct.

It was claimed that removal of area 4 alone produced flaccid paralysis (removal of excitation), while removal of 6 alone, and especially 6 *plus* 4 (removal of inhibition), caused spastic paralysis. Damage in the internal capsule produced either type according to the kind of fibres affected. Damage to the pyramidal tract below the pons produced only flaccid paralysis, suggesting that the inhibitory fibres had now passed off into the cerebellum to influence the rubro-spinal tract, leaving only the excitatory fibres in the pyramid.

There was no evidence to support the view that the basal ganglia represented an "old motor pathway".

On the evolutionary scale cortical representation appeared to have extended, firstly, to include more and more of the body, and secondly, to reach smaller and smaller muscle units, so permitting a progressively finer control. It might be assumed that area 6, which was chiefly concerned with complete movements, integrated the excitatory capacity of area 4 with the inhibitory capacity of area 4s to produce the coordinated movements which were characteristic of stimulation of area 6.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 124 and 129, of July 11 and 18, 1946.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Emergency List.

Transfer to Retired List.—Surgeon Captain Alexander Scott-Mackenzie is transferred to the Retired List, dated 26th June, 1946.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

To be Surgeon Commanders.—Surgeon Lieutenant-Commanders John Kempson Maddox and James Aloysius Feodius Flynn.

AUSTRALIAN MILITARY FORCES.

Australian Army Medical Corps.

The notification respecting NX208067 Captain B. C. Mendelsohn which appeared in Executive Minute No. 113 of 1946, promulgated in *Commonwealth Gazette* No. 97 of 1946, is amended to read under the above heading.

70th Australian Camp Hospital.—The notification respecting NX208062 Captain L. F. Breitner which appeared in Executive Minute No. 113 of 1946, promulgated in *Commonwealth Gazette* No. 97 of 1946, is amended to read under the above side heading.

WX1546 Colonel (Temporary Brigadier) G. B. D. Maitland, D.S.O., D.C.M., relinquishes the appointment of Deputy Director-General of Medical Services, "A" Branch, Army Headquarters, and is placed upon the Regimental Supernumerary List, 10th May, 1946.

NX108193 Lieutenant-Colonel (Temporary Colonel) C. R. Cole relinquishes command of 101st Australian General Hospital (Australian Imperial Force) and is placed upon the Regimental Supernumerary List, 10th May, 1946.

VX14845 Lieutenant-Colonel N. M. Eadie is appointed to command 101st Australian General Hospital (Australian Imperial Force), 10th May, 1946.

NX12614 Lieutenant-Colonel C. R. B. Blackburn, WX3469 Major (Temporary Lieutenant-Colonel) S. L. Mainland, O.B.E., and NX35096 Lieutenant-Colonel E. C. Palmer, O.B.E., are placed upon the Regimental Supernumerary List, 10th May, 1946.

SX34115 Captain I. G. Pavy is placed upon the Regimental Supernumerary List, 27th April, 1946.

VX13622 Major (Temporary Lieutenant-Colonel) A. D. Matheson relinquishes the rank of Temporary Lieutenant-Colonel and is transferred to the Reserve of Officers (Australian Army Medical Corps), 27th April, 1946.

NX474 Lieutenant-Colonel M. S. Alexander, O.B.E., is transferred to the Reserve of Officers (Australian Army Medical Corps), 27th April, 1946.

1st Australian Out-Patients' Depot.—NX200337 Captain W. N. C. G. Bennett is placed upon the Regimental Supernumerary List, 21st March, 1946.

VX39198 Lieutenant-Colonel A. E. Coates is transferred to the Reserve of Officers (Australian Army Medical Corps), 6th December, 1945.

Inter-Service Medical Wing Demobilization Centres (Australian Military Forces Component): To be Temporary Major, 7th May, 1946.—NX34727 Captain W. R. Davison.

Reserve of Officers.

The undermentioned officers are transferred to the Reserve of Officers with effect from the dates indicated, and, where applicable, cease to be seconded. Officers holding temporary rank relinquish such temporary rank on the date of transfer to the Reserve of Officers.

Majors VX14457 R. Warden, 1st May, 1946, and QX40818 T. R. Biggs, 24th April, 1946, and SX22320 Captain A. P. Cherry, 2nd May, 1946.

No. 102 (Holland Park) Military Hospital.—Captains QX22735 R. Hertzberg, 24th April, 1946, and QX38337 B. N. Adsett, 1st May, 1946.

No. 105 (Adelaide) Military Hospital.—SX9262 Major R. Motteram, 1st May, 1946.

No. 110 (Perth) Military Hospital.—WX29065 Major A. M. Clarke, 20th March, 1946, and WX11108 Captain H. C. Mulcahy, 1st May, 1946, and WX32015 Major T. M. Gilbert, 3rd May, 1946.

No. 113 (Concord) Military Hospital.—NX107908 Major T. F. Rose, 2nd May, 1946, and Captains NX112019 M. C. Seton, 1st May, 1946, and NFX135722 G. U. Grogan, 24th April, 1946.

No. 115 (Heidelberg) Military Hospital.—VX55182 Major J. I. Hayward, 1st May, 1946.

121st Australian General Hospital (Australian Imperial Force).—SX27742 Major R. N. C. Bickford, 2nd May, 1946.

2nd Australian Camp Hospital.—QX40890 Major T. K. Durbridge, 25th April, 1946.

20th Australian Camp Hospital.—VX138579 Captain A. C. Jeffrey, 1st May, 1946.

102nd Australian Convalescent Depot.—QX25551 Captain A. W. Eklund, 18th April, 1946.

107th Australian Convalescent Depot.—SX15735 Major M. C. Newland, 30th April, 1946.

102nd Australian Casualty Clearing Station (Australian Imperial Force).—NX160740 Major T. J. Constance, 24th April, 1946.

104th Australian Casualty Clearing Station (Australian Imperial Force).—NX70254 Major V. H. Vernon, 19th April, 1946.

109th Australian Casualty Clearing Station (Australian Imperial Force).—NX100988 Major H. K. Uren, 25th April, 1946.

110th Australian Casualty Clearing Station (Australian Imperial Force).—SX11115 Major J. W. Sangster, 7th May, 1946.

Inter-Service Medical Wing Demobilization Centres (Australian Military Forces Component).—Captains QX57387 F. H. Cowlishaw, 18th April, 1946, NX169487 B. F. Gates, 4th May, 1946, NX77339 R. A. G. Holmes, 19th April, 1946, NX77287 H. I. McKenzie, 30th April, 1946, and VX133202 C. H. B. Adamson, 1st May, 1946, and WX3357 Major D. W. Macpherson, 1st May, 1946.

QX901 Lieutenant A. J. Clark, 5th December, 1945, and VX14906 Captain J. R. Goding, 30th April, 1946.

The rank of VX39055 Lieutenant-Colonel H. A. Phillips is as now shown and not as it appeared in Executive Minute No. 84 of 1946, promulgated in *Commonwealth Gazette* No. 78 of 1946.

2nd/2nd Australian General Hospital.—VX117079 Captain (Temporary Major) W. D. Curtis, 9th May, 1946.

No. 102 (Holland Park) Military Hospital.—QX59519 Captain G. A. Ruscoe, 3rd May, 1946.

No. 110 (Perth) Military Hospital.—TX6419 Captain T. J. K. Jamieson, 7th May, 1946.

No. 115 (Heidelberg) Military Hospital.—VX14867 Major D. G. Duffy, 9th May, 1946, and VX69797 Captain T. O. Beatty, 7th May, 1946.

121st Australian General Hospital (Australian Imperial Force).—Captains S43063 F. G. T. Turner and SX29059 L. C. Lum, 10th May, 1946.

4th Australian Field Ambulance.—WX31099 Captain T. E. Waters, 8th May, 1946.

Inter-Service Medical Wing Demobilization Centres (Australian Military Forces Component).—VX28211 Major P. J. Parsons, 7th May, 1946.

The undermentioned officers are transferred to the Reserve of Officers with effect from the dates indicated, and, where applicable, cease to be seconded. Officers holding temporary rank relinquish such temporary rank on the date of transfer to the Reserve of Officers, and are granted from such date honorary rank on the Reserve of Officers equivalent to the temporary rank relinquished.

NX70929 Captain (Temporary Major) W. F. L. Liggins, 24th April, 1946.

118th Australian General Hospital (Australian Imperial Force).—WX3417 Captain (Temporary Major) D. L. Mercer, 27th April, 1946.

2nd/3rd Australian Field Ambulance.—NX71094 Captain (Temporary Major) J. R. O. Roger, 3rd May, 1946.

Retired List.

The undermentioned officers are placed upon the Retired List on the dates indicated, and, where applicable, cease to be seconded. Officers holding temporary rank relinquish such temporary rank on the date of placement upon the Retired List.

Captains VX70316 W. L. Sloss, 30th April, 1946, VX90842 F. A. L. Bacon, 27th April, 1946, and VX94716 K. J. Lipshut, 2nd May, 1946.

No. 112 (Brisbane) Military Hospital.—QX35638 Captain J. H. Crawford, 30th April, 1946.

No. 113 (Concord) Military Hospital.—VX133227 Captain N. C. Lee-Tet, 1st May, 1946.

No. 115 (Heidelberg) Military Hospital.—VX108252 Major P. G. Dowling, 27th April, 1946.

11th Australian Field Ambulance.—NX103432 Captain J. W. H. Mulhearn, 24th April, 1946.

118th Australian General Hospital (Australian Imperial Force).—QX45117 Captain A. R. McGregor, 18th January, 1946.

Post-Graduate Work.

THE MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

PROGRAMME FOR SEPTEMBER.

Course for F.R.C.S. Part I.

CLASSES designed for candidates for the Part I F.R.C.S. (England) examination to be held in Melbourne early in January, 1947, have been arranged by the Melbourne Permanent Post-Graduate Committee. The course, which will commence on September 2 and continue on Monday and Wednesday afternoons, is as follows:

Anatomy and Histology.—Approximately 25 lecture demonstrations, 12.45 to 2.15 o'clock p.m., Mondays and Wednesdays. Fee: 10 guineas.

Applied Physiology and the Principles of Pathology.—Approximately 30 lectures, 2.30 to 4 o'clock p.m., Mondays and Wednesdays; and approximately 15 demonstrations, 4 to 5 o'clock p.m., Wednesdays. Fee: 20 guineas.

It is assumed that the student will commence the course with a knowledge of anatomy and advanced physiology. This assumption is necessary because of the limited time available. Tuition in pathology and physiology will be confined to the study of pathology from the physiological point of view. To this end a series of suitable topics and demonstrations has been selected.

It is anticipated that copies of questions set at recent examinations will soon be available.

Although these classes are primarily designed for those preparing for F.R.C.S. Part I, they are available to any registered medical practitioner who desires a presentation of these subjects.

Applications from those proposing to attend these classes should be forwarded to the Post-Graduate Committee's office by August 21. Service medical officers entitled to financial assistance under the Commonwealth Reconstruction Training Scheme part time are requested to indicate this fact on their application. It would be appreciated if the fee for the classes could accompany the application from those not so entitled.

Continuous Refresher Course.

Medical and surgical classes for service and ex-service medical officers will be continued on four days a week throughout the month at the various hospitals and the Pharmacy School. A syllabus of subjects has been published.

Gynæcology and Obstetrics Refresher.

The fourth special course for supernumeraries at the Women's Hospital will commence on August 26 and run till September 21.

Course at Geelong.

Dr. J. W. Johnstone will give a demonstration on "Endocrines" at the Geelong Hospital on September 18 at 8.30 o'clock p.m. Those wishing to attend should communicate with Dr. Alan Kidd, 216, High Street, Belmont, Geelong.

Enrolments for Courses.

Enrolments for courses in Melbourne should be made with the Secretary of the Committee, College of Surgeons, Spring Street, Melbourne, C.1. Telephone: JM 1547-8.

Correspondence.

A METHOD FOR THE REMOVAL OF TONSILS AND ADENOIDS UNDER LOCAL ANÆSTHESIA WITH THE PATIENT IN THE RECUMBENT POSITION.

SIR: In my article on tonsillectomy in the recumbent position under local anæsthesia, published in the issue of June 22, 1946, I stated that Dr. D. G. Perrett had been the instigator. Dr. Perrett did tell me that he had used this method, but for how long I am not sure. Since the publication of the article I have been informed that Dr. A. O. Davy demonstrated a similar method at a meeting of the Australasian College of Surgeons in 1938. In addition, Dr. D. Ada, Dr. P. McIndoe, Dr. E. Hedberg and Dr. A. O. Davy had used a similar method prior to my arrival in the Middle East.

When I was working with Dr. Davy in the Middle East, he and his assistant were using the recumbent position, but had a nurse holding a tongue depressor instead of using the Boyle Davis gag.

I think there is no doubt that to Dr. Davy goes the credit for being the initiator of this method.

Dr. Perrett might be interested enough to state when he first removed tonsils in this way.

Yours, etc.,

R. H. BETTINGTON.

Craignish,
185, Macquarie Street,
Sydney.
July 20, 1946.

THE RED CROSS REHABILITATION FARM, "GILBULLA".

SIR: I would like to draw the attention of all medical men to the rehabilitation farm which has been established by the Australian Red Cross Society at Menangle, about forty miles from Sydney. This farm, named "Gilbulla", was established in November, 1944, as a residential centre for men of this war who have been discharged from the services after medical treatment, and find that through some disability, nervous or physical, they are unable for a time to resume normal community life.

Recommendations for admission are received from service medical officers or from civilian doctors, and there is a medical advisory committee which is responsible for the acceptance of the cases. The society's social workers, before admission is arranged, obtain facts concerning the man's personal and family responsibilities, and are in a position to offer help when anxiety in this direction might prevent their taking the opportunity of going to "Gilbulla". This help covers financial arrangements.

A medical practitioner visits the farm regularly each week accompanied by a social worker, and before admission each man has explained to him the objects of the farm and what will be required of him in planning for his rehabilitation. Under medical supervision each man is given graduated work to help him regain normal working capacity, and it is particularly stressed that the ultimate aim is permanent employment or vocational training on discharge. To this end, regular visits are made by representatives of the Commonwealth Employment Service, Department of Post-War Reconstruction, and Army Rehabilitation Service, so that each man has had the opportunity to make definite plans for his future before leaving "Gilbulla".

It is the opinion of the Medical and Management Committees that "Gilbulla" should not be regarded as an institution for men with serious mental disabilities, and that every effort should be made to exclude those men who have chronic mental disorders. To this effect, a time limit is imposed on a person's stay at the farm, and it has been found that this helps the men's attitude in effecting a quick recovery.

The following groups of men are considered suitable for admission to "Gilbulla".

1. Ex-servicemen suffering from neurosis who are not making a satisfactory adjustment in the occupational, domestic or social spheres. These men should be given a trial in civilian life for at least three months before admission to "Gilbulla" is suggested.
2. Remitted psychotics soon after discharge from service.
3. Ex-prisoners of war who are debilitated or "flabby-fat".
4. Ex-prisoners of war who are lacking in confidence as to their ability to work and with no definite plan for civilian occupation.
5. Ex-servicemen convalescent from acute illness, medical or surgical, who would not require any nursing care and who would be likely to benefit from the physical reconditioning of farm life.
6. Ex-prisoners of war who generally require help in settling back to normal life.

Applications from any State for admission may be lodged with the Social Service Departments of the Red Cross, which are situated in the capital city of each state.

Yours, etc.,

J. NEWMAN MORRIS,
Chairman, Australian Red Cross Society.

122-128, Flinders Street,
Melbourne,
June 25, 1946.

THE NATIONAL HEALTH SERVICE BILL OF GREAT BRITAIN.

SIR: In reply to the letter of Dr. Dane in the issue of July 6, I would like to remind Dr. Dane that some hundred years ago it was an amazing and unheard of thing to suggest that all men should have equal opportunities and rights. We have become accustomed to speaking of freedom in many things, and of the various rights of an individual. I am not alone in my opinion that one of man's most sacred rights is the right to happiness and health. Dr. Dane may accept or reject this idea, but if accepted he must follow it through to a conclusion. If it is the right of every man to enjoy happiness and health, every modern scientific advance should be directed towards the achievement of this right. Every citizen plays his part in society according to his ability, education and environment. Some may appear to contribute more than others, for which I would blame society as a whole. In return, every citizen is entitled to receive what we may term his rights. Are we going to continue the practice of requiring every citizen to buy what should be his by right—the means of achieving happiness and health? The medical men should act as the dispensers, not the sellers, of health.

Dr. Dane is undoubtedly wise when he questions the word "free", because after all nothing can be completely "free" in any form of community life. But surely Dr. Dane realizes that the word was used in the same sense as when it is applied to education, justice and defence. Considering defence alone, while it is easy and understandable to assume that defence against invasion is the concern of every member of the community and is financed, not by every member paying for his own defence, but by the community as a whole, defence against disease is considered an entirely different question. And to continue the example, when we need to keep an army of men fit and able to fight we inaugurate a medical service which is comprehensive and available to all, but when there is no war, we do not consider the civilian worthy of being kept fit and able to live decently; in fact we only consider health from a national point of view when we are at war; in peace it is nobody's concern. From time to time, as private practice has shown its inability to fulfil the community's needs in various branches of health, we find them incorporated in the various public health departments, for example, sanitation, quaran-

tine, infectious disease, care of children and mothers and so on.

Just as the strength of a chain depends on its weakest link, so the well-being of the community as a whole depends on the health of every individual. It is therefore in the interest of the community that the health of every member should be safeguarded as a national responsibility. Any health must be comprehensive and available without qualification to all. In assessing the basic wage only a small proportion is allowed for health purposes. The cost of any health service, if it is to fulfil these requirements, cannot be borne equally by all, and it must be the higher income groups who after all receive a greater proportion of Australia's goods, who must contribute most through their taxation to the national health. Such a service, while not "free" in the ultimate sense, would not place on the individual the strain of financial embarrassment in times of illness and would be a positive contribution towards the achievement and maintenance of health.

Yours, etc.,

STEFANIA SIEDLECKY.

The Rachel Forster Hospital,
Redfern,
New South Wales.
July 18, 1946.

SPINAL ANÆSTHESIA AND CHLOROFORM: A COMPARISON OF MORTALITY.

SIR: I have read Dr. Marshall's letter in your issue of July 20 on the subject of myself and my iniquity in standing up and daring to reply stiffly to criticism passed by him and Dr. Thomas on an article of mine. It was a study of facts. They were proper subjects for study. There is at present no control over inquiry. It is free, and I was free to choose my subject without limitation or licence from Dr. Marshall or anyone who may abhor "dangerous thought". In my reply, I had to protect myself against mischievous and perverse misquotation, foolish charges, and barbed advice, and I did it. It is now sufficiently obvious that my defence was successful. I did not expect it to be pleasing to my critics. It was not pleasing.

Free and undirected, my subject was the over-all results of spinal anæsthesia, all cases, all methods, all administrants, and comparison with similar results of chloroform anæsthesia. The article was factual and statistical. The figures taken were the only ones of their kind that were known, or at any rate known to me. They alone had the necessary character of random distribution, and so they alone could give the necessary data for a statistically sound inquiry. The article was a cold study of cold figures.

My crime consisted in making the study, finding out the figures, and telling, whereas one of my critics expressed the opinion that they would have been better buried.

I ventured to say that when deaths occurred under spinal anæsthesia, default on the part of the practitioner was not necessarily the cause, though it could be. I gave reasons for saying this, and I expressed the belief that "skill is not everything", which means that deaths can occur without blame being attachable to anyone.

In my defence I recalled that Lawrie and the Hyderabad Commission reported over fifty years ago that chloroform could be given in every case "with perfect ease and perfect safety" if it was given properly, and that Lawrie had supported this by quoting a series of 45,000 cases without a death. It was at once seen by critics that this meant that every death that occurred must be the fault of the practitioner, and Lawrie himself explicitly confirmed it as meaning that.

Looking back now, it seems to me to cast some doubt on the infallibility of human opinion, at all events when it is opinion about anæsthesia.

But Dr. Marshall is undeterred. In both his first and second letters he has taken the same line about spinal anæsthesia as was taken in those former times about chloroform. Every death must be the fault of the practitioner. In his most recent letter he tells us that "subject to suitable precautions, however (for example, control of ascent, serial blood pressure readings, use of vaso-pressors and oxygen), it may be applied in cases of doubtful and even poor tolerance with almost absolute safety". In a comment on my remark that "skill is not everything", he indulges in declamatory rhetoric like this: "It is astonishing that a surgeon of the standing and experience [of myself] should be at such pains to discount the value of skill in the use of anæsthesia, whether general or spinal. This is an incomprehensible absurdity, which any informed person will reject with the contumely it deserves." *Ignoratio elenchi!* Some of us, perhaps, have read enough logic to recognize this ancient trick.

I say again, skill, knowledge and care will save lives, but not every life, for deaths will occur even when there is no lack of skill or care.

In short, Dr. Marshall is wrong. If Lawrie was not infallible fifty years ago, neither is Dr. Marshall today. But if he is right, we have arrived at the point that, if a death occurs, and an inquest follows, the proper verdict for the jury should be one condemning the practitioner. Call Dr. Marshall to the witness box. Cross-examine him. Listen to him. You know what he has written. You know what he must say.

This letter may end here by asking where ether stands if spinal analgesia can be applied "with almost absolute safety" in those bad cases referred to. Deaths do occur under ether, and I am sure they do not always signify incompetency. I have seen several, and I would not blame anybody for them. Should we then go to spinal anæsthesia for the "almost absolute safety" obtainable in such parts of the body as are anatomically suitable for it?

Yours, etc.,

C. E. CORLETTE.

Sydney,
July 22, 1946.

CONGENITAL FIBROCYSTIC DISEASE OF THE PANCREAS.

SIR: I wish to thank Dr. Margaret Harper for her letter appearing in your issue of July 20, 1946, on congenital fibrocystic disease of the pancreas.

Dr. Harper states: "In none of the articles in the American literature that I have seen has there been an adequate description of the stools, the authors contenting themselves with noting the occurrence of 'diarrhœa'." I therefore refer her again to H. B. Philipsborn, G. Lawrence and K. C. Lewis in the *Journal of Pediatrics*, Volume XXV, 1944, in which she will find a description of the very variable types of stools noted by investigators abroad.

Dr. Harper mentions that a child receiving a high protein diet with little fat may pass stools having a putrefactive odour. Consequently I would ask Dr. Harper whether she considers the appearance of the stools is, or is not, dependent on the type of food which has been used? Again, it would be interesting to know whether liquid fat can be seen in stools containing only 12% and even 3·3% of unsplit fat—in the latter case, at least, it must be a most delicate observation.

In cases in which the fæcal fat is largely in the split form it would seem unlikely that anything more than a "presumptive" diagnosis could be made from inspection of the stools. It is here that the estimation of tryptic activity in the duodenum is useful, obviating the necessity for post-mortem confirmation of the diagnosis and enabling an accurate and certain prognosis to be made.

Yours, etc.,

P. E. BLAUBAUM.

111, Collins Street,
Melbourne,
July 26, 1946.

Public Health.

CANCER SURVEY IN NEW SOUTH WALES.

THE following statement is published at the request of Dr. A. B. Lilley, chairman of the Hospitals Commission of New South Wales.

In connexion with the Government's intention to erect a Cancer Institute in Sydney, and in order to obtain an indication of the incidence and types of cancer occurring in country districts, arrangements have been made for four consultants, a surgeon, gynæcologist, dermatologist and radiotherapist to visit country districts.

The first visit will be made to Goulburn and Wagga, the consultants being in Goulburn on September 9 and 10 and in Wagga on September 11 and 12, 1946.

The consultants desire to see all cancer patients in hospitals, all doubtful cases in which they may assist in the diagnosis, and all positive cases in which local practitioners may desire to discuss methods of treatment. It is desirable that the maximum number of cases possible be brought forward.

The work of the consultants will be carried out in an honorary capacity, and it is their desire to see any patient the practitioner may wish, whether private or public.

The consultations will be carried out at the Goulburn District and Wagga Base Hospitals, which will make all the necessary arrangements. The cooperation of the medical practitioners in the Goulburn and Wagga districts is therefore sought in order to make the survey successful.

It is suggested that they communicate with the hospitals regarding details as to reception of patients *et cetera*.

Australian Medical Board Proceedings.

SOUTH AUSTRALIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1919 to 1935, of South Australia, as duly qualified medical practitioners:

Ball, Karl George, M.B., B.S., 1946 (Univ. Adelaide), 63, Park Terrace, Parkside.
Condon, Robert Francis, M.B., B.S., 1946 (Univ. Adelaide), 210A, Young Street, North Unley.
Coats, Douglas Alan, M.B., B.S., 1946 (Univ. Adelaide), 140, Wakefield Street, Adelaide.
Moore, Max Clifford, M.B., B.S., 1946 (Univ. Adelaide), 10, Bagot Avenue, Mile End.
McCann, Ralph Newton, M.B., B.S., 1946 (Univ. Adelaide), 52, Tusmore Avenue, Tusmore.
McCann, William James, M.B., B.S., 1946 (Univ. Adelaide), 52, Tusmore Avenue, Tusmore.
Cheesman, Graham Frank, M.B., B.S., 1946 (Univ. Adelaide), Springbank Road, Clapham.
Beard, Roland Maurice Charles Georges, M.B., B.S., 1946 (Univ. Adelaide), 6, Nottage Terrace, Medindie Gardens.
Nancarrow, Malcolm Ewart, M.B., B.S., 1946 (Univ. Adelaide), 15, Stanley Street, Woodville.
Haselgrove, Harold Frederick, M.B., B.S., 1946 (Univ. Adelaide), 33, Stepney Street, St. Peters.
Fisher, Anthony Graham, M.B., B.S., 1946 (Univ. Adelaide), 8, Second Avenue, St. Peters.
Faunce, Marcus de Laune, M.B., B.S., 1946 (Univ. Adelaide), 42, Glenunga Avenue, Glenunga.
Emery, Warren Frederick, M.B., B.S., 1946 (Univ. Adelaide), 56, West Terrace, Kensington Gardens.
Nicholls, Jack Montague, M.B., B.S., 1946 (Univ. Adelaide), 85, Findon Road, Woodville.
Scragg, Roy Frederick Rhodes, M.B., B.S., 1946 (Univ. Adelaide), 27, Prospect Terrace, Prospect.
Venner, Barton Frederick, M.B., B.S., 1946 (Univ. Adelaide), 16, Herbert Road, Ashford.
Wyllie, Geoffrey Gurner, M.B., B.S., 1946 (Univ. Adelaide), 59, Burnside Road, Kensington Park.
Fisk, Graham Bristow, M.B., B.S., 1946 (Univ. Adelaide), 25, Watson Avenue, Rose Park.
Pash, Hannah June, M.B., B.S., 1946 (Univ. Adelaide), 21, Hilda Terrace, Hawthorn.
Seppelt, Yvonne Granville, M.B., B.S., 1946 (Univ. Adelaide), 43, Glenunga Avenue, Glenunga.
Hayward, George Murray, M.B., B.S., 1943 (Univ. Melbourne), Mount Gambier.
Hawke, Vivian Lee, M.B., B.S., 1946 (Univ. Adelaide), 28, Unley Road, North Unley.
Trembath, Frank Everard, M.B., B.S., 1944 (Univ. Adelaide), 142, Esplanade, Largs.
Gowland, John Hudson, M.B., B.S., 1930 (Univ. Melbourne), 248, Orrong Road, Toorak, Victoria.
Roxburgh, Russell, M.B., B.S., 1943 (Univ. Sydney), Wudinna.
Roxburgh, *née* Graham, Phyllis Mary, M.B., B.S., 1943 (Univ. Sydney), Wudinna.
Kohler, Theodor George, M.B., B.S., 1946 (Univ. Adelaide), 50, South Terrace, Adelaide.
Martin, Stanley Bamford, M.B., B.S., 1946 (Univ. Adelaide), 31, Glenunga Avenue, Glenunga.
Bonnin, James Arthur, M.B., B.S., 1946 (Univ. Adelaide), 48, Mills Terrace, North Adelaide.
Sibthorpe, Gwenyth, M.B., B.S., 1946 (Univ. Adelaide), 30, Main Street, Henley Beach.
Russell, Ronald Edyvane, M.B., B.S., 1946 (Univ. Adelaide), 19, Parker Street, Mile End.
Bennett, Donald Collier, M.B., B.S., 1946 (Univ. Adelaide), 4, West Linton, Tarlton Street, Somerton.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts*, 1939 to 1940, of Queensland, as duly qualified medical practitioners:

Cassidy, Desmond John, M.B., B.S., 1944 (Univ. Sydney), c.o. Dr. Williams, Mitchell.
Grant, John Halford, M.B., B.S., 1946 (Univ. Queensland), General Hospital, Brisbane.
Rimmer, *née* Bell, Betty Jeanette, M.B., B.S., 1945 (Univ. Sydney), Lowood.
Branster, Russell, M.B., B.S., 1945 (Univ. Sydney), General Hospital, Brisbane.
Droulers, John Paul Maurice, M.B., B.S., 1944 (Univ. Sydney), General Hospital, Bundaberg.
Katz, Irene, M.B., B.S., 1946 (Univ. Queensland), McLeod Street, Herston, Brisbane.
Makinson, Jill, M.B., B.S., 1946 (Univ. Sydney), General Hospital, Rockhampton.

Research.

WYETH FELLOWSHIP IN CLINICAL MEDICINE.

WYETH INCORPORATED have donated a fellowship in clinical medicine to the Walter and Eliza Hall Institute of Medical Research. The first Fellow to be appointed is Dr. Peter J. Parsons, a graduate of medicine and science at the University of Melbourne. Dr. Parsons will work as a member of the Clinical Research Unit which has recently been formed to carry out clinical research in the wards of the Royal Melbourne Hospital and the adjoining laboratories of the Walter and Eliza Hall Institute. Dr. Parsons, who has recently been discharged from the Australian Army Medical Corps after six years' service, will reside in the Royal Melbourne Hospital during the term of his appointment, which is for one year.

Obituary.

HAROLD WHITRIDGE DAVIES.

DR. A. J. CANNY has sent the following appreciation of the late Professor Harold Whitridge Davies.

Harold Whitridge Davies, known affectionately as "Pete", was appointed Professor of Physiology in the University of Sydney in 1930, and held that appointment until his recent sudden death. In that period of nearly sixteen years he brought into the university an understanding of the worth of many elusive but most important things, of those intangible elements which are the essence of true culture; the love of learning for its own sake and not for the material gain to be garnered from its prostitution; the joy of tinkering with the clockwork of the universe just to see how it works without a thought for the publicity or the advancement which it might bring to the tinkerer; the sensual delights of music, of colour and of poetry, and the spiritual ecstasy of a good deed done for a fellow man. These concepts wither and die too readily in our harsh antipodean world, or assume grotesque distorted forms. They did not perish or become misshapen under "Pete" Davies's care and tutelage. To his staff and to many of his students he spread the divine contagion which he himself had experienced as the pupil of Dr. J. S. Haldane in schools of physiology at Oxford.

Thanks to his efforts, there was at long last established a chair of biochemistry in the University of Sydney, and had his advice been taken there would ere this have been created a chair or at least a readership in pharmacology. He tried to bring the organization of the medical sciences into line with European standards; a lesser man would have striven to retain within his own grasp the reins of petty power which as Professor of Physiology he might in accordance with tradition have claimed to hold.

Without considering his own interests he fought for the rights of his staff and students, and with characteristic generosity sought for them opportunities which without his encouragement they would have thought unattainable.

"Pete" Davies loved the world and life, the broad heavens and the open road. Whatever faults he had were as the imperfections of nature, without malice and without envy, patent for all to see and unhidden by any cloak of pious and hypocritical rectitude. No fault of his ever hurt his fellow

men. Most generous indeed he was; generous above all in that he put the interests of others before his own.

When we think of him in the days to come we will measure his achievement, not by the sum of his scientific publications alone, but by the help and inspiration which he gave to others, by the breadth of his kindness and generosity and by the depth of his sincerity and friendship.

LESLIE GORDON DAVIDSON.

WE regret to announce the death of Dr. Leslie Gordon Davidson, which occurred on July 12, 1946, at Cremorne, New South Wales.

VERA SCANTLEBURY-BROWN.

WE regret to announce the death of Dr. Vera Scantlebury-Brown, which occurred on July 14, 1946, at Melbourne.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Austin, John Charles, provisional registration, 1946 (Univ. Sydney), 53A, Raglan Street, Mosman.

Silberstern, Ernest, provisional registration, 1946 (Univ. Sydney), Waterfall Sanatorium, Waterfall.

Fennell, Thomas Joseph, M.B., 1939 (Univ. Sydney), Flat 3, 7, Shadforth Street, Mosman.

Hicks, Zoe Isobel, M.B., B.S., 1945 (Univ. Sydney), 41, O'Connor Street, Haberfield.

Williams, Geoffrey Athol, provisional registration, 1946 (Univ. Sydney), District Hospital, Lithgow.

THE FEDERAL MEDICAL WAR RELIEF FUND.

THE following contributions to the Federal Medical War Relief Fund have been received:

Queensland.

Downs and South-Western Local Association, £113 7s.

R. B. Charlton, Milton Geaney, H. D. Ashton, D. Gifford Croll, J. W. Heaslop, £10 10s.

Duncan Fowles, I. C. Hains, A. W. Graham, E. T. C. Schmidt, M. L. D. McKeon, R. M. Smith, £5 5s.

Humphrey C. Lloyd, £2 2s. 6d.

C. J. Murphy, Joyce Stobo, Eleanor E. Bourne, £2 2s.

Anonymous, £1 1s.

Total: £206 16s. 6d.

Grand total: £14,087 5s. 6d.

Notice.

ON the night of July 18, 1946, a motor-car was stolen in Murwillumbah, New South Wales, and amongst its contents was a Brown Buerger cystoscope, a Watson-Victor monocular microscope and a folding enamel operating table. Any practitioner who may be offered these articles is asked to communicate with the police.

Books Received.

"Child Treatment and the Therapy of Play", by Lydia Jackson, B.Sc. (Oxon.), and Kathleen M. Todd, M.B., D.P.M., with an introduction by Emanuel Miller, M.A., M.R.C.P., D.P.M.; 1946. London: Methuen and Company Limited. 8½" × 5½", pp. 125. Price: 8s. 6d.

"The Surgical Technic of Abdominal Operations", by Julius L. Spivack, M.D., LL.D.; Fourth Edition Revised; 1946. Springfield: Charles C. Thomas. 9¾" × 6¾", pp. 728, with many illustrations. Price: $10.00.

"In Crocodile Land: Wandering in Northern Australia", by Ion L. Idriess; 1946. Sydney and London: Angus and Robertson Limited. 8½" × 5½", pp. 262, with many illustrations. Price: 8s. 6d.

"Ancient Anodynes: Primitive Anæsthesia and Allied Conditions", by E. S. Ellis, M.R.C.S., F.R.A.I., with Foreword by T. K. Penniman, M.A.; 1946. London: William Heinemann (Medical Books) Limited. 8½" × 5½", pp. 192. Price: 21s.

"Morell Mackenzie", by R. Scott Stevenson; 1946. London: William Heinemann (Medical Books) Limited. 8½" × 5½", pp. 202, with illustrations. Price: 15s.

"Hermaphroditos: The Human Intersex", by A. P. Cawadias, O.B.E., M.D., F.R.C.P.; Second Edition; 1946. London: William Heinemann (Medical Books) Limited. 8½" × 5½", pp. 91, with many illustrations. Price: 15s.

"Sanitary Science Notes: A Handbook for Students", by H. Hill, F.R.San.I., F.S.I.A., A.M.I.S.E., and E. Dodsworth, M.R.San.I., M.S.I.A.; 1946. London: H. K. Lewis and Company Limited. 6¾" × 4¼", pp. 143. Price: 7s. 6d.

"Occupational Therapy for the Limbless", by Phyllis Lyttleton, C.S.P., M.A.O.T.; 1946. London: H. K. Lewis and Company Limited. 7¼" × 4¾", pp. 48, with many illustrations. Price: 3s.

Diary for the Month.

AUG. 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.
AUG. 7.—Western Australian Branch, B.M.A.: Council Meeting.
AUG. 7.—Victorian Branch, B.M.A.: Branch Meeting.
AUG. 8.—South Australian Branch, B.M.A.: Council Meeting.
AUG. 9.—Queensland Branch, B.M.A.: Council Meeting.
AUG. 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
AUG. 13.—Tasmanian Branch, B.M.A.: Ordinary Meeting.
AUG. 20.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2).

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such a notification is received within one month.

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